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# THE OPHTHALMOLOGIST AND THE LIGHTING PROBLEMS OF HIS PATIENTS

WALTER B. LANCASTER, M.D.

BOSTON

What knowledge of illumination may be expected of the ophthalmologist? Poor lighting may be divided, for the purposes of this paper, quite arbitrarily into two classes. In the first the lighting is so defective that no expert knowledge is required to condemn it. In the second group the lighting is defective, but its defects are not obvious; and quite frequently the user of the light and the dealer who supplied the fixtures and the manufacturer who made them, and even a large corporation like the General Electric Company who advertises them may think the arrangement is good. Patients using such defective lighting often say that they have the best there is. It may fairly be expected of the ophthalmologist that he should be able to recognize defective lighting of the second type, since it is frequently a cause of his patient's symptoms. He should know enough about the principles involved to be able to convince those whom he advises, to be able to give reasons and not simply opinions, since somebody's opinions are likely to be quoted on the other side of the argument.

Most ophthalmologists probably have their own more or less established ideas about lighting, certainly about the kind they prefer themselves. Standards have changed: What was deemed very good lighting twenty-five years ago would be classed second or third rate today. Ophthalmologists ought to know what modern illuminating engineers have made available, and ought to make use of this improved practice.

Poor lighting of the first, grossly defective type will produce symptoms in the best and strongest of eyes. Poor lighting of the second type, the type that often passes for good lighting, does not produce much effect on strong healthy eyes. Only prolonged use extending over many months will cause such eyes to show symptoms, more especially when some contributing factors are breaking down the general resistance. On the other hand, given eyes that are in some way defective, poor lighting is just the thing to bring out the symptoms of these defects. Thus errors of refraction in themselves will cause conjunctival irritation

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and headache in many people, but they are much helped in causing these symptoms if the eyes are used under poor illumination.

The symptoms that lead me to expect that a patient is using his eyes with poor illumination are those of conjunctival irritation not attributable to other causes such as infection (conjunctivitis), nor to mechanical or chemical irritants such as dust, smoke, fumes, wind, sun, etc., nor to the most common of all these causes, errors of refraction and heterophoria. Besides the scratchy, dry, itching, sandy, burning sensations, perhaps with photophobia, there are often more severe symptoms, fatigue and headache.

When confronted with a problem of lighting, it will be found useful to analyze it under the following heads:

(*a*) The quantity, intensity or *brightness.*

(*b*) The quality, color or *wavelength.*

(*c*) The *distribution,* whether direct and concentrated, or indirect and diffuse; and especially whether there is *glare.*

(*a*) Many tests have been made to determine the amount of light needed for good seeing. Books like Clark's [1] "Lighting in Relation to Public Health" and Luckiesh and Moss' [2] "Seeing" give accounts of many of these with references.

When the objects looked at are black on white, a light of 10 foot-candle power gives nearly maximum visual acuity. This means that the surface looked at has a brightness of about 10 millilamberts if of good reflecting power.[3] If the brightness is increased to 100 ml., the eye sees a very little better. Somewhere in the vicinity of 5,000 ml. the acuity decreases. It is clear that for such objects as Snellen charts or well printed books, 10 ft. c. is sufficient for very good vision. With 10 ft. c., a white surface has a brightness of nearly 10 ml.

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1. Clark: Lighting in Relation to Public Health, Baltimore, Williams & Wilkins, 1924.

2. Luckiesh, M., and Moss, F. K.: Seeing: A Partnership of Lighting and Vision, Baltimore, Williams & Wilkins, 1931.

3. Illuminating engineers have found it necessary in the interest of exact thinking to coin technical terms. The light from a source is measured in candles; the standard is the international candle. The light falling on a surface is measured in lumens = luminous flux; a foot-candle is equal to 1 lumen per square foot. The light given off by a surface, that is, the brightness of a surface, is measured in lamberts; the millilambert is more convenient. A millilambert is about equal to an "apparent foot-candle." Hence, if a source of 10 candle-power shines on a surface 1 foot square, 1 foot away, the flux of light is 10 lumens, and the surface brightness will depend on how good a reflector it is. If perfect, the brightness would be 10 millilamberts, 10 apparent foot-candles. If the surface were only a moderately good reflector, say 50 per cent, then the brightness would be 5 millilamberts. Evidently the millilambert is very important, for it measures the amount of light that comes to the eye from whatever surface is being looked at.

(*b*) When tests are made involving speed, it is found that an increase above 10 ml. (up to 20 or even 50 ml.) results in a moderate improvement.

(*c*) When tests are made involving different degrees of contrast—that is, when instead of black letters on a white background, gray letters on a gray background are used—the results are very important. To understand them something must be known about the way the brightness of a surface (which is measured in millilamberts) depends not only on the intensity of the illumination falling on the surface or object, but on the coefficient of reflection of that surface or object.

## QUANTITY

The important thing is the amount of light that comes to the eye from the object at which one is looking, compared with the light from the surrounding field. If the object is a piece of dark cloth with some faint spots on it, the amount of light coming to the eye can be computed if one knows (1) the amount of light incident to the cloth (suppose this to be 10 ft. c.) and (2) the coefficient of reflection of the cloth and of the spots (suppose this to be 10 per cent for the cloth and 12 per cent for the spots). If the cloth reflected 100 per cent, its brightness would be 10 ml. As it reflects only 10 per cent of the light falling on it, its brightness under an illumination of 10 ft. c. is 1 ml. The brightness of the spots is 1.2 ml., and the contrast is 20 per cent; that is, the spots are 20 per cent brighter than the cloth. The contrast is sufficient for rather easy seeing, but the brightness is far too low. It should be about ten times as much for the best seeing. *To get 10 ml. of brightness from a surface of only 10 per cent reflecting power needs an illumination of 100 ft. c.*

Therefore, never think of lighting solely in terms of the intensity of the source, but always think of the properties of the objects to be looked at. If it is a white paper with black letters, that is one thing; if it is dark cloth or black leather with dark stitches on it, that is quite another.

The calculation of the maximum, minimum and optimum amount of light for different kinds of work in different factories, shops, etc., can be left to the engineer. One should know in a general way that 10 ft. c. is ample for reading, but that fine work, especially on poorly reflecting objects with little contrast and under conditions requiring speed, needs from two to ten times as much.

Is there any danger of too much light? There is often complaint that the artificial light is too bright; but nearly always the fault is in the distribution which produces a *glare*. It is enlightening to compare the intensity or brightness under average conditions at midday out of

doors. The brightness is over 1,000 ft. c. In the direct sunlight it is from 5,000 to 10,000 ft. c. Obviously the eyes tolerate a brightness of over 1,000 ft. c. without discomfort if the conditions are favorable; that is, if the distribution of the light is satisfactory, and if the eye is in a suitable state of adaptation. It is a shock to change quickly from one intensity to a very different one. Contrast with background out of doors is rarely over 20 to 1.

### QUALITY, THAT IS, WAVELENGTH OR COLOR

The eye was evolved to work under daylight, hence it is natural to assume that light of daylight quality is the best for all conditions. It is undoubtedly the best for some conditions. But both experience and theory teach that for many conditions something different from daylight is very good, sometimes better than daylight. The eye finds light agreeable that is deficient in the ends of the spectrum, especially the blue. This is true, for example, for reading. Thus the Mazda electric light is well adapted for reading and the like. In my opinion, it is superior to the blue-bulb daylight lamp. On the other hand, if artificial light has to be used for work involving color matching, it should be of daylight quality. There are special conditions where monochromatic light gives the best results. For example, the greenish mercury vapor lamp is superior for some purposes; again, red-free light is valuable for detecting some retinal lesions. These are special conditions easily understood. For the vast majority of cases the ordinary gas-filled incandescent lamp is extremely satisfactory as far as quality (wavelength) goes. *When results of a given system of lighting are not satisfactory, it is usually a matter of distribution of the light, or of brightness of the lighting, rather than of quality (wavelength).*

### DISTRIBUTION

The eye dislikes direct concentrated lighting from a source of high intrinsic brilliancy, with intense shadows, and an extreme range between the light part and the dark part of the room. The eye is more efficient and more comfortable with diffuse light, such as is found in a room well lighted by daylight, or in a room well lighted by indirect artificial light.

Intrinsic brilliancy is the brightness per unit area—candles per square centimeter. The sky is about equal in brightness per square centimeter to the flame of a candle. The incandescent filament is a thousand times as bright per square centimeter. It is important to avoid high intrinsic brilliancy. This is done by indirect lighting or by placing sources such as incandescent or arc lamps inside of reflectors or shades or large diffusing globes, so that the actual source is not visible; the surface of

the globe becomes the source. If it is not feasible to do this, the lights should be so placed that they are at least 30 degrees above the line of sight of the worker so that they will shine on the periphery of the retina, not on the macula. When the image of a source of high intrinsic brilliancy falls on the periphery of the retina, though it is disagreeable, it is much less so than when near the macula.

About twenty-five years ago a committee was appointed by the Section on Ophthalmology of the American Medical Association to report on the relative merits of direct and indirect lighting. This was not long after indirect lighting had been introduced. The committee invited Professor Ferree to help in the solution of this question. During a series of years Ferree and Rand carried on their studies of this problem, and showed convincingly that indirect lighting was both more comfortable to the eyes and enabled them to do more work. Many others have written on the subject, and it can be considered a well established fact. This does not mean that there is no field for direct lighting, or for a combination of the two. Bear in mind that I said in the beginning that good, strong, healthy eyes can tolerate lighting that is not the best. Ophthalmologists are consulted by patients because their eyes are defective or troublesome. For these patients one must be prepared to point out what is the kind and amount of light that will give ***them*** the least trouble. If indirect lighting is advised, it should be pointed out that it is like daylight in being diffuse, free from strong shadows and free from glare, but that it is not satisfactory unless there is enough of it. When indirect lighting was first tried for draughting, the men were much dissatisfied and demanded additional local light on the table. Ten years later someone tried it again, and the men were greatly pleased and satisfied with it. The difference was that at the first trial twenty-five years ago, the light supplied was 2½ apparent foot candles on the table; the second trial started with 15 ft. c. and increased to 17 ft. c. or more. Undoubtedly more than 17 would be advised today.

To get adequate brightness by indirect lighting, it is a great help if the walls and ceiling are very good reflectors, either white or cream white, or light buff—not bluish or any dark color. A room with poorly reflecting walls and ceiling may easily require five or ten times as many watts to light it as would be the case if it were finished with white. Ordinarily the present standard calls for from 1 to 3 watts per square foot. For example, a room 20 by 15 feet would require from 300 to 1,000 watts, depending, of course, on the kind of work to be done in the room, as well as on the color of walls and ceiling.

How the indirect lighting is to be installed is a matter for the engineer and dealers. It is enough to point out that it is not necessary to buy expensive fixtures. Under many conditions lamps are deemed nec-

essary for esthetic reasons. These can be made to supply a large flux of light on the ceiling and upper part of the walls by having a reflector hidden by the shade. The reflector throws the light upward instead of downward. Similarly, wall brackets can be provided with small reflectors, throwing the light upward, and concealed by shades of any type of beauty that appeals to the owner. When an indirect or semi-indirect luminaire is used, it is important that the reflector throw the light not in a small area on the ceiling, a common fault, but over the whole ceiling and down on the walls as far as the top of the doors.

The common bridge-lamp, which is a type in wide general use, is not an indirect light, but the light is directed down on to the table and the players. It does well enough for strong eyes, but is far from being the best kind of light for playing cards or other games. For reading, etc., it is not good practice to have the light so arranged that there is bright light on the book or work, while the surrounding field is dim. A similar error is made by those who have their test charts lighted by, say, 20 ft. c. (a good standard), but have the field around the chart relatively dark. It has been proved over and over again that the eye does ordinary work better when the surrounding field is lighted to an intensity not widely different from the work itself, not much darker, and not much lighter. The use of gray operating sheets and gowns, and gray walls in the operating room as high as the operator's head, arose from a knowledge of this fact. Most persons think that when they arrange a lamp to throw a good strong light on the book, while the surrounding room is in relative darkness, they have done the best that is possible for their eyes, especially if they take care to have the light come over the left shoulder.

All this is a relic of the days when our ancestors had to depend on a kerosene lamp, a student lamp, or an Argand gas burner. It was not feasible to light the whole room. With modern, diffuse, general, good illumination, one can sit anywhere in the room and see well. Library reading rooms and study rooms in colleges and private schools are rarely well lighted. Here is an opportunity for good propaganda.

Glare is excessive brightness of the whole or any part of the field of vision, sufficient to cause reduced vision, fatigue or discomfort.

Glare is the most common defect of artificial lighting. Out of doors a contrast exceeding 1 to 20 between an object and its background is rare. Indoors it is not rare to find a contrast from ten to one hundred times as great; for instance, a bare or even a frosted electric lamp against a rather dark wall. If direct lighting is used, and there are frequent conditions where some direct lighting is desirable, the lamp itself should be concealed from view of the worker at his work. This is done by placing it behind or above the worker or inside of a reflector or shade.

If a light is so placed that it is reflected from the paper into the eyes, as by a mirror, the result is glare by reflection. If the paper is shiny, this is greatly increased; but if the light is diffuse, there is almost no glare by reflection.

To minimize glare, have no exposed sources of high intrinsic brilliancy. If the source (for example, a semi-indirect luminaire) is not much over twenty times as bright as its background, there will not be much glare. If there must be exposed sources, have them over 30 degrees above the line of sight. Contrast can be reduced by increasing the brightness of the background. An automobile headlight at night against a dark background is intensely glaring; by day against a light background, the glare may be hardly noticeable. Avoid a location of the source that will favor glare by reflection, for example, a desk lamp in front of the worker, reflecting from the paper or from a glass-topped desk into the worker's eyes. The time of exposure is a factor. An exposed light falling on the periphery of the retina hour after hour is more annoying than a brief exposure.

The average patient thinks that the best lighting is obtained by a desk lamp, reading lamp or bed lamp provided with a shade or a reflector that throws the light directly on the paper or book, while the rest of the room is relatively dark. In many cases it is sufficient to tell the patient that such arrangements, while good enough for strong, well eyes, are not suitable for weak, sensitive eyes, and to advise general lighting of the whole room, preferably by indirect light with very much more powerful bulbs.

In other cases it is necessary to go into the matter in more detail. If the patient is intelligent enough, he will wish to know some of the reasons, and he will be more likely to cooperate if he is convinced. I find that a good starting-point is daylight. Point out that in a room well lighted by daylight, the whole room is lighted, and that the eye does not adapt itself comfortably to a bright light on the book or paper while the rest of the room is dark. The eye prefers diffuse light without sharp contrasts and dense shadows. This is well illustrated by my penumbra test. A pencil held parallel to a sheet of paper and 3 or 4 inches away will show by the shadow it makes on the paper whether the light is diffuse, coming from all directions, or concentrated, coming from a small intense source. If there is a window with good daylight, the pencil will cast a hardly distinguishable, broad, blurry shadow that is all penumbra. If the shade is drawn and a bare electric light used, the pencil will cast a sharp clear shadow with hardly any penumbra. With a good indirect or even a semi-indirect light, the shadow will be like that from the window light, indistinct, all penumbra. If this is demonstrated to the patient, he can easily repeat it at home or office and

show whether his light is good diffuse light, or concentrated, harsh, trying light.

Next point out that when indirect light is not found satisfactory, it is usually because there is not enough of it. The usual standards are from 1 to 3 watts per square foot of floor space. A room 15 by 15 feet would need 250 watts if the walls and ceiling were light, 500 to 750 watts if the walls were dark.

## SUMMARY

It is my purpose in this paper to point out that while much of the poor lighting in use is so obviously defective as to be recognizable without expert knowledge, the important thing for the ophthalmologist is to be able to recognize the poor lighting which is not obvious, which in fact is often deemed good or even very good, but is nevertheless the cause of the symptoms for which the patient has consulted him, symptoms which persist after treatment by glasses and by collyria.

Such patients require better than ordinary good lighting. It is necessary to imitate daylight lighting by having the whole room lighted with general diffuse light. Avoid local concentrated light of high intrinsic brilliancy, even if it is shaded from the eyes; have the lighting soft and free from sharp shadows and strong contrasts. Be sure that there is enough light for the kind of work to be done. If it is black and white, like reading, 10 ft. c. is enough. This requires about 1 watt per square foot in a room with light ceiling and walls, if indirect lighting is used. If it is gray on gray, or any work of small contrasts and low coefficient of reflection, 2 or 3 watts per square foot are needed for indirect lighting, sometimes supplemented by additional direct lighting when conditions require it.

Finally, lighting that is good enough for good, strong, healthy eyes is quite inadequate for eyes that are sick, or sensitive or strained.

# CIRCULATION OF THE AQUEOUS

## II. MECHANISM OF REABSORPTION OF FLUID

JONAS S. FRIEDENWALD, M.D.

AND

H. F. PIERCE, PH.D.

BALTIMORE

In a previous paper,[1] we showed how the Leber bubble manometer may be used to estimate the rate of circulation of the aqueous. The present study represents the further application of the same technic to the measurement of the rate of escape of fluid from the anterior chamber under various conditions

The present state of our knowledge in regard to the mechanism by which the aqueous is reabsorbed into the circulating blood leaves much to be desired. The older notion that the fluid leaks out through actual pores in the walls of Schlemm's canal has been disproved by injections of colloids and suspensoids into the anterior chamber. Duke-Elder [2] showed that the hydrostatic pressure in the vessels connected with Schlemm's canal is normally above the intra-ocular pressure, so that the conception of simple filtration has had to be abandoned. No evidence has ever been discovered, in spite of numerous attemps, to show that fluid is removed from the anterior chamber by an active secretion into the lumina either of Schlemm's canal or of the vessels of the iris. We are, therefore, reduced to a consideration of the possible rôle which osmotic or other nonmechanical forces may play in this process.

As we have shown, the rate of flow of aqueous is small in comparison to the rate of diffusion of dissolved substances into and out of the aqueous. It may be assumed, therefore, that the pressure within most of the intra-ocular vessels is such as to nearly balance the difference in osmotic pressure between the fluid without and within. This equilibrium level is, no doubt, slightly exceeded in the ciliary capillaries, while the pressure in the vessels concerned with reabsorption of fluid must be slightly below this equilibrium value. It has been shown that when the pressure within the ocular vessels is raised, by raising the general arterial or venous blood pressure, a similar change in the intra-ocular pressure

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From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

1. Friedenwald, J. S., and Pierce, H. F.: The Circulation of the Aqueous: I. Rate of Flow, Arch. Ophth. **7**:538 (April) 1932.

2. Duke-Elder, W. S.: The Ocular Circulation, Brit. J. Ophth. **10**:513, 1926.

results. It is true that the change in the intra-ocular pressure is generally small in comparison with the change in blood pressure, and that the intra-ocular pressure generally returns to normal before the blood pressure returns to normal. The return to normal is usually more complete in the face of increased arterial pressure than of increased venous pressure. This discrepancy has been attributed to some unknown compensating mechanism. The present study represents an attempt to explain this discrepancy in terms of the normal dynamics of the reabsorptive process.

Similarly, it has been shown that an increase in the permeability of the intra-ocular capillaries with partial destruction of the osmotic barrier between plasma and aqueous results in the formation of so-called plasmoid aqueous. If, for instance, a solution of histamine is injected into the vitreous of an experimental animal, plasmoid aqueous is formed. We showed [3] that, under these circumstances, if the intra-ocular pressure is held constant, an abnormally rapid formation of aqueous takes place, while, on the other hand, if no new channel for its escape is provided, an increased intra-ocular pressure results. These considerations have been applied to the understanding of the pathogenesis of acute glaucoma and of some forms of glaucoma secondary to uveitis. The paradoxical fact remains, however, that in many instances of intra-ocular inflammatory disease in which the vessels are congested, the tissues edematous and the aqueous plasmoid, and in which, therefore, there is every reason to assume that an abnormally rapid formation of aqueous is taking place, nevertheless, the intra-ocular pressure may remain at its normal level or even fall below normal.

In animal experimentation the analogy to this paradox is to be found in the injection of histamine into the anterior chamber, which, as we have already reported, is followed by a fall in intra-ocular pressure and an increased rate of reabsorption of aqueous. In spite of this fall in pressure, the iris is found to be hyperemic and edematous, and the aqueous contains large quantities of plasma. Here again we are forced to suppose that some compensating mechanism exists, whereby, in the face of need, an increased rate of reabsorption of fluid takes place.

The compensating mechanism which we require to explain these phenomena may be attributed either to the simple dynamics of the reabsorptive process, or to the interference of higher control by the central nervous system through vasomotor reflexes. The experiments that we are reporting have led us to advance a purely mechanical explanation of these phenomena. We have no doubt, however, that this mechanism is subject to the control of vasomotor reflexes which may retard or enhance its operation. Nevertheless, there are some grounds for believing,

3. Friedenwald, J. S., and Pierce, H. F.: The Pathogenesis of Acute Glaucoma, II. Experimental Study, Arch. Ophth. 5:574 (May) 1930.

*a priori,* that a purely mechanical explanation lies at the bottom of these processes. In the first place, all of the anomalies that we are called on to explain are characterized by congestion of the vessels in the anterior segment of the globe. In the second place, neither patients in whom the ophthalmic branch of the fifth nerve has been cut, nor those who have suffered paralysis or resection of the cervical sympathetic nerves appear to be robbed of this compensating mechanism. They show no special predisposition to glaucoma as a complication of iritis or corneal ulcer. These two nerves contain all of the vasomotor fibers that are known to innervate the intra-ocular vessels. Undoubtedly these patients often show some disturbance in their intra-ocular pressure, but the compensating mechanism in which we are particularly interested is not necessarily abolished.

## RATE OF REABSORPTION OF AQUEOUS UNDER INCREASED PRESSURE

The first step in the study of this problem consisted in the measurement of the rate of reabsorption of fluid out of the eye at various pressures above the normal level. Under sodium barbital anesthesia, after instilling a few drops of 1 per cent cocaine into the conjunctival sac, a cannula was introduced into the anterior chamber of a dog's eye. The cannula was connected with the modified Leber bubble manometer described in the previous paper. At first the reservoir of the manometer was adjusted to balance the intra-ocular pressure. Then the reservoir was raised by intervals of 5 or 10 cm. and the flow of fluid into the eye during periods of from five to ten minutes was measured by the movement of the bubble in the capillary tube. At each change of pressure a considerable displacement of fluid into the eye took place due to the distention of the sclera and the expulsion of some blood from the intra-ocular vascular bed. This readjustment to a higher pressure level takes place almost instantaneously. In order to avoid complicating our readings by this factor, the movement of the bubble during the first minute after a change in pressure was disregarded. Thereafter, the movement of the bubble toward the eye was found to be reasonably uniform, minute for minute, though showing, of course, periodic variations associated with the cardiac and respiratory cycles.

At pressures only slightly above the normal level, the measured volume of flow cannot be considered with certainty as representing the total amount of fluid absorbed from the eye during that period. An unknown fraction of the normal circulation must be added to the measured amount. However, the magnitude of the flow measured at even a few millimeters of mercury pressure above the normal intra-ocular level was generally many times as great as the normal flow. The error due to this cause is, therefore, quite small.

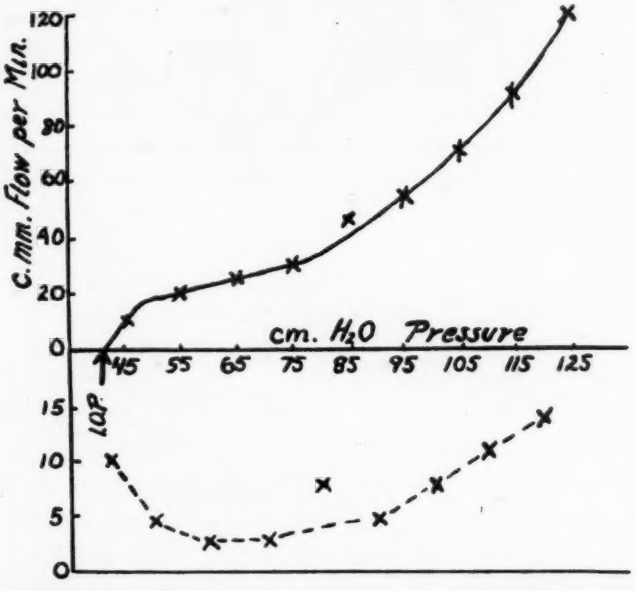


Chart 1 (exper. 1).—Effect of intra-ocular pressure on the rate of reabsorption of fluid from the anterior chamber. The curve *x*——*x* indicates the flow versus intra-ocular pressure. The curve *x*...*x* represents the increment in the rate of reabsorption of fluid for each 5 cm. water increment in pressure.

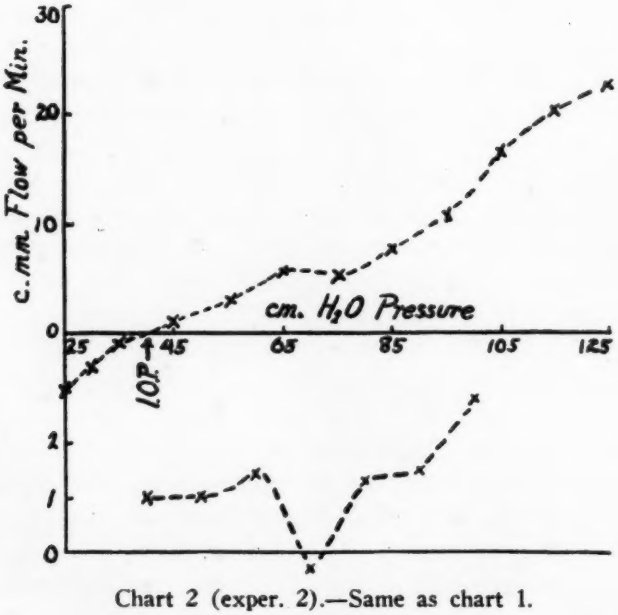


Chart 2 (exper. 2).—Same as chart 1.

If one charts the results of such an experiment, plotting rate of flow versus pressure (charts 1 and 2), one obtains a curve which shows certain very interesting characteristics. It consists in three segments, an initial sharp rise, a plateau and a second rise. The first segment of the curve represents the effect of pressure changes in the immediate neighborhood of the normal pressure. Here the flow increases rapidly with slight increase in pressure. A pressure of not more than 5 mm. of mercury above the normal intra-ocular is generally associated with a rate of absorption of fluid of from 5 to 10 c.mm. per minute, i. e., from five to ten times the normal circulation. It is not without interest to know

TABLE 1.—*Data in Experiment 1*

| Water Pressure, Cm. | Time, Minutes | Flow, C.Mm. | Flow per Minute, C.Mm. |
|---|---|---|---|
| 40 | .. | 0 | 0 |
| 45 | 8 | 85 | 10.6 |
| 55 | 5 | 99 | 19.8 |
| 65 | 5 | 126 | 25.2 |
| 75 | 8 | 242 | 30.7 |
| 85 | 8 | 371 | 46.4 |
| 95 | 4 | 220 | 55.0 |
| 105 | 6 | 424 | 70.7 |
| 115 | 4 | 370 | 92.5 |
| 125 | 8 | 968 | 118.0 |

TABLE 2.—*Data in Experiment 2*

| Water Pressure, Cm. | Time, Minutes | Flow, C.Mm. | Flow per Minute, C.Mm. |
|---|---|---|---|
| 25 | 8 | —41 | —5.0 |
| 30 | 7 | —21 | —3.0 |
| 35 | 7 | —8 | —1.1 |
| 45 | 9 | 8 | 0.9 |
| 55 | 9 | 27 | 3.0 |
| 65 | 9 | 42 | 5.7 |
| 75 | 7 | 36 | 5.1 |
| 85 | 9 | 68 | 7.6 |
| 95 | 11 | 117 | 10.6 |
| 105 | 9 | 147 | 16.3 |
| 115 | 9 | 183 | 20.3 |
| 125 | 7 | 159 | 22.7 |

that the mechanism for the reabsorption of aqueous has reserve powers of this magnitude within what may be considered as a normal range of pressure variation. The end of this first segment of the curve was found in various experiments between 3 and 10 mm. of mercury above the normal intra-ocular pressure.

The second segment of the curve is the plateau, the region where a change in pressure produces only the slightest change in the rate of flow. The flow may even decrease slightly as the pressure rises. The plateau usually extends up to a pressure of from 20 to 25 mm. of mercury above the normal intra-ocular pressure. At this point a second bend occurs in the curve and further increases in pressure usually cause considerable increase in the rate of flow. Each of the following experiments represents one of several similar experiments in which essentially identical results were obtained.

EXPERIMENT 1 (July 3, 1930).—A male dog weighing 9.15 Kg. was used for the experiment. Sodium barbital, 4.1 Gm., was injected intravenously. The mean arterial blood pressure was 120 mm. of mercury throughout the experiment (chart 1).

EXPERIMENT 2 (Nov. 4, 1930).—A male dog weighing 14.6 Kg. was used. Sodium barbital, 6.5 Gm., was injected intravenously. The mean arterial blood pressure was constant throughout the experiment. The cannula was introduced into the left anterior chamber (chart 2).

Before attempting to interpret the significance of the three segments of this curve, we are forced to decide whether the flow measured represented the almost exclusive action of a single vascular bed within the eye, or whether several tissues were concerned in the process. The existence of rates of reabsorption of comparable magnitude in two partially independent vascular beds might account for the sinuosity of our curves.

TABLE 3.—*Data on Experiment 3*

| Time, P.M. | Intra-ocular Pressure, Cm. of $H_2O$ | Distance from Cornea to Lens in Enlarged Photographic Image, Mm. |
|---|---|---|
| 4:08 | 35 | 6.5 |
| 4:09 | 35 | 6.1 |
| 4:15 | 65 | 6.3 |
| 4:17 | 65 | 6.2 |
| 4:47 | 65 | 6.2 |
| 5:17 | 65 | 6.4 |
| 5:47 | 65 | 6.3 |
| 6:18 | 65 | 6.0 |
| Mean | | 6.25 |
| Average deviation from the mean | | 0.12 |

## THE OUTFLOW OF FLUID FROM THE POSTERIOR SEGMENT OF THE GLOBE

In order to discover what fraction of the reabsorption of fluid takes place in the segment of the globe behind the iris, the following experiment was performed:

EXPERIMENT 3 (April 2, 1931).—A female dog weighing 6.5 Kg. was used. One-fourth grain (16 mg.) of morphine was given subcutaneously, followed in one-half hour by 2.2 Gm. of sodium barbital intravenously. A cannula was inserted into the anterior chamber of the left eye, and was connected with a reservoir of physiologic solution of sodium chloride which was raised to a level 25 cm. higher than that required to balance the normal intra-ocular pressure. A flow into the eye of from 15 to 20 c. mm. per minute was obtained. A narrow beam of light was so arranged as to pass down the optical axis of the eye, and a camera was set at 45 degrees to this axis. The whole system, including the dog's head and eye, was held rigid during the course of the experiment. Photographs of the optical section of the cornea and lens by the slit-lamp beam were taken at intervals of thirty minutes. In this way any change in depth of the anterior chamber greater than 0.04 mm. could be clearly registered. If under these circumstances any fluid is withdrawn from the vitreous, a corresponding amount of fluid runs into the anterior chamber from the reservoir and a backward movement of the iris-lens diaphragm is seen.

The photographs were so taken as to give an image of the same size as the object, and were enlarged 4.4 times. Measurement of the apparent depth as seen at a 45 degree angle were then made.

No consistent change in depth of the anterior chamber greater than the probable error of measurement was found. In order to reduce these measurements to the actual size, we divide by 4.4 (the magnification of the image) and multiply by $\sqrt{2}$ to correct for the 45 degree angle of view (omitting correction for the refractive effect of the cornea). We thus arrive at the value $\frac{0.12}{4.4} \times \sqrt{2} = 0.038$ mm. as the probable error of the measurements in actual depth of the anterior chamber.

The volume of aqueous in the anterior chamber involved in this error is equal to that of a cylinder having this height and the plane of the iris and pupil as its base:

$$v = d\ \pi\ r^2 = 0.038 \times 3.14 \times 36 \text{ c. mm.} = 4 \text{ c. mm.}$$

Though the experiment was allowed to run for two hours, no measurable change in depth of the anterior chamber was observed. Providing, therefore, that the iris-lens diaphragm remained water tight during this period, we can conclude that not more than 4 c. mm. of fluid disappeared from the posterior portion of the eyeball. During the same period, over 2,000 c. mm. of fluid was absorbed from the anterior chamber.

The possibility of a leak through the iris-lens diaphragm was tested by a similar experiment in which a mixture of potassium ferrocyanide and iron ammonium citrate solutions was allowed to flow into the anterior chamber instead of salt solution. At the end of two hours, the eye was removed and fixed in acid formaldehyde, which caused a deposit of prussian blue wherever the solution had penetrated. The anterior chamber and the tissue surrounding it including the pupillary zone of the lens were found to be deeply stained with the precipitate, but the posterior chamber contained only the faintest trace of blue. We conclude, therefore, that even under extreme conditions of pressure, the reabsorption of aqueous by the tissues behind the iris in a dog's eye is inappreciable in amount.

*Comment.*—Returning now to the interpretation of the three segments of the curve representing aqueous absorption versus pressure, it is first of interest to note that the pressure level at which the plateau first appears corresponds to that which Duke-Elder [2] found characteristic of the intra-ocular veins, while the end of the plateau corresponds to the pressure level at which he estimates that of the intra-ocular capillary system. Using these data, we make the following working hypothesis to explain our findings. We suppose that the main reabsorption of aqueous is accomplished through Schlemm's canal and the veins of the iris. Under normal circumstances, the pressure in these vessels is slightly above the intra-ocular pressure. Absorption of aqueous takes place in virtue of the excess of the difference in osmotic pressure

between the fluid within and without these vessels, over the difference in hydrostatic pressure within and without. It is not necessary to assume that the canal of Schlemm is normally filled with serum. Since the pressure within the canal is probably the lowest of all these venous channels, it can hardly exceed the intra-ocular pressure by more than 1 or 2 mm. of mercury. The protein osmotic pressure of plasma, on the other hand, exceeds that of the aqueous by from 20 to 25 mm. of mercury. At equilibrium between hydrostatic and osmotic forces, therefore, the plasma within the canal may be diluted with as much as fifteen times its volume of aqueous. Any excess over this low concentration of plasma would be sufficient to attract additional fluid from the anterior chamber into the canal.

A slight rise in intra-ocular pressure would, according to this hypothesis, be expected to increase considerably the rate of absorption of fluid, since such a rise, up to equality in pressure within and without the venous channels, would add to the effectiveness of the mechanism of osmotic absorption. This is the course of events which we conceive as taking place during the first, sharply rising arm of our curve.

If the intra-ocular pressure is suddenly raised above that of these veins, a momentary stoppage of venous blood flow must occur, after which with rising venous pressure, due to the *vis a tergo,* most of the channels should reopen. The relation between intra-ocular pressure and intra-ocular venous pressure should, in this way be constantly reestablished on the same basis throughout the whole range over which compensating readjustment of venous pressure can take place. This would explain the plateau in our curves.

At the upper end of the plateau, the curve has a sharp break and from here onward maintains a smooth and rapid ascent which continues without a break far beyond the arterial blood pressure level. It would appear, therefore, that the escape of aqueous during this portion of the curve occurs by extra vascular channels, possibly through the perivascular tissue spaces of the anterior perforating vessels.

### EFFECT OF INCREASED VENOUS PRESSURE

It is a corollary of this conception that in the region of the plateau not all the available vascular channels are patent. Those which normally lie farthest down the pressure gradient should be most easily occluded. This conclusion furnishes a possible experimental test of our hypothesis, for, if the hypothesis is correct, an increase in general venous pressure should make possible the reopening of these occluded channels and increase the rate of absorption of aqueous, a result which under other circumstances would be regarded as paradoxical. The following experiment was performed.

EXPERIMENT 4 (Nov. 11, 1930).—A female dog, weighing 10.1 Kg. was used. One-fourth grain of morphine was given subcutaneously followed in one-half hour by 3.5 Gm. of sodium barbital intravenously. Tracheotomy was performed.

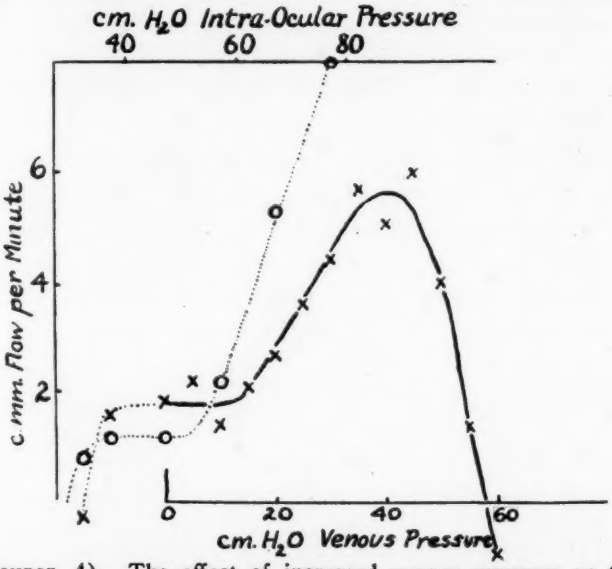


Chart 3 (exper. 4).—The effect of increased venous pressure on the rate of reabsorption of fluid from the anterior chamber. The curve *O...O* indicates the flow versus intra-ocular pressure obtained on the left eye; *x...x*, the flow versus intra-ocular pressure obtained on the right eye; *x——x*, the flow versus venous pressure obtained on the right eye, with the intra-ocular pressure held stationary.

TABLE 4.—*Data in Experiment 4*

| | Intra-ocular Pressure, Cm. $H_2O$ | Venous Pressure, Cm. $H_2O$ | Time, Minutes | Flow, C.Mm. | Flow per Minute, C.Mm. |
|---|---|---|---|---|---|
| Left eye..... | 32 | 0 | 9 | 7 | 0.8 |
| | 37 | 0 | 9 | 11 | 1.2 |
| | 47 | 0 | 9 | 11 | 1.2 |
| | 57 | 0 | 17 | 38 | 2.2 |
| | 67 | 0 | 9 | 48 | 5.3 |
| | 77 | 0 | 9 | 72 | 8.0 |
| Right eye.... | 32 | 0 | 10 | —3 | —0.3 |
| | 37 | 0 | 11 | 18 | 1.6 |
| | 47 | 0 | 9 | 17 | 1.9 |
| | 47 | 5 | 13 | 29 | 2.2 |
| | 47 | 10 | 8 | 11 | 1.4 |
| | 47 | 15 | 10 | 21 | 2.1 |
| | 47 | 20 | 9 | 24 | 2.7 |
| | 47 | 25 | 10 | 36 | 3.6 |
| | 47 | 30 | 10 | 44 | 4.4 |
| | 47 | 35 | 9 | 51 | 5.7 |
| | 47 | 40 | 10 | 51 | 5.1 |
| | 47 | 45 | 10 | 60 | 6.0 |
| | 47 | 50 | 10 | 40 | 4.0 |
| | 47 | 55 | 10 | 14 | 1.4 |
| | 47 | 60 | 10 | —9 | —0.9 |

The blood pressure was recorded from the right femoral artery. The cannula was inserted into the left anterior chamber and the flow recorded at various levels of pressure above the normal intra-ocular pressure. The cannula was

then withdrawn from the left eye and inserted into the right anterior chamber. The same procedure was repeated on the right eye, but when the intra-ocular pressure had reached the region of the plateau in the flow versus intra-ocular pressure curve, the intra-ocular pressure was not changed further. The cuff of a clinical blood pressure apparatus was now placed about the dog's neck, and the pressure in this cuff raised by gradual stages. The rate of flow into the eye at stationary intra-ocular pressure by changing venous pressure was recorded (chart 3).

A possible objection to the interpretation that we have given to this experiment lies in the suggestion that, with venous stasis, histamine-like substances may accumulate in the intra-ocular tissues resulting in vasodilatation, and possibly in a change in permeability that is due to chemical irritation rather than a direct result of intravascular pressure. To test this possibility, we made the following experiment.

EXPERIMENT 5 (Jan. 21, 1931).—A female dog weighing 9.3 Kg. was used. Anesthesia was produced as in the previous experiments. Three-tenths cubic

TABLE 5.—*Data in Experiment 5*

| Substance | Wheal | Substance | Wheal |
|---|---|---|---|
| Histamine solution 1:3,000 | +++ | Histamine solution 1:3,000 | +++ |
| Histamine solution 1:30,000 | + | Histamine solution 1:30,000 | + |
| Histamine solution 1:300,000 | ± | Physiologic solution of sodium chloride | 0 |
| Physiologic solution of sodium chloride | 0 | Aqueous II | 0 |
| Aqueous I | 0 | Aqueous I | 0 |

centimeter of aqueous was removed from the left eye (aqueous I). One drop of this fluid, together with drops of physiologic solution of sodium chloride and of varying strengths of histamine solution, was placed on the skin of a human subject and minute pricks were made through these drops with a sharp needle. The results are shown in table 5. A blood pressure cuff was then put around the dog's neck and inflated to a pressure of 21 cm. of water. After the cuff had been in position for one hour, aqueous was withdrawn from the right eye (aqueous II), and compared with the previously obtained specimen (aqueous I), and with salt solution and histamine solutions. The results show that histamine is not present in the anterior chamber either before or after venous stasis in amounts equal to the minimum which we have previously shown necessary to produce a change in the rate of reabsorption of the aqueous.

*Comment.*—The results thus far enable us to formulate a purely mechanical explanation of the compensating mechanism which is partially successful in reducing to normal the intra-ocular pressure which has been raised through increased venous pressure. The primary effect of a rise in venous pressure in the head is, as so many writers have demonstrated, a rise in intra-ocular capillary pressure, an increased rate of formation of aqueous and an increased intra-ocular pressure. A secondary effect is an increased rate of reabsorption of aqueous with a tendency to reduce the intra-ocular pressure toward normal.

### EFFECT OF INCREASED ARTERIAL PRESSURE

A second corollary to be drawn from the hypothesis regarding the mechanism of reabsorption of aqueous which we have outlined is that a rise of arterial pressure should cause an increase in the rate of reabsorption of aqueous both at normal intra-ocular pressure and at the pressure level of the plateau, the former in virtue of a more rapid flow of plasma into Schlemm's canal, the latter through increased venous pressure reopening obstructed venous channels. These effects should be most marked if the rise in arterial pressure is associated with peripheral vasodilatation. On the other hand, an increased arterial and capillary pressure within the eye should cause an increased rate of formation of fluid. The available methods of experiment enable us to measure only the difference between these two opposing effects, and it was to be expected that the increased rate of reabsorption of aqueous, if it existed, would in most cases be masked by an equal or larger increase in the rate of formation of the fluid. We were fortunate, however, in being able to render the effect on the rate of reabsorption demonstrable in a few instances.

EXPERIMENT 6 (Oct. 22, 1931).—A male dog weighing 8 Kg. was used. Anesthesia was produced as in the previous experiments. Laparotomy was performed, and a loose ligature was placed under the abdominal aorta just below the renal arteries. The ends of the ligature were brought out through the incision, through a glass tube the end of which rested loosely on the aorta. The loose ends of the ligature were clamped in a hemostat. The arrangement was such that the ligature could be wound onto the hemostat and tightened like the strings of a violin, pulling a knuckle of the aorta into the tube and occluding its lumen. Continuous arterial blood pressure tracings were then taken from the brachial artery. A cannula was inserted into the left anterior chamber connected through the bubble manometer with a reservoir of salt solution which was raised to a level 40 cm. above the eye. A second cannula was inserted into the right anterior chamber connected with a compensating small bore recording mercury manometer. After a period of ten minutes, during which a steady state, in flow, intra-ocular pressure and arterial blood pressure was attained, the ligature about the aorta was tightened, giving an immediate rise in arterial pressure. After five minutes, the mean arterial pressure began to fall. This fall was associated with an increase in the size of the pulse pressure which continued to the end of the experiment; the mean arterial pressure, however, never went below the steady level it had shown prior to the occlusion of the aorta. The intra-ocular pressure, in the right eye, followed symmetrically the initial rise in arterial pressure, but during the period of vasodilatation fell below its previous level. The flow into the left anterior chamber from the stationary reservoir at 40 cm. of water pressure stopped momentarily at the outset of the rise in arterial pressure, and then gradually increased reaching finally twice its initial velocity (chart 4).

The application of these results to the explanation of the return of the intra-ocular pressure to normal in the face of an increased arterial pressure requires no further elaboration.

TABLE 6.—*Data on Experiment 6*

| Time | Flow from Reservoir at 40 Cm. $H_2O$ Pressure Into Left Eye, C.Mm. $H_2O$ per Minute | Intra-ocular Pressure Right Eye, Cm. $H_2O$ | Mean Arterial Blood Pressure, Mm. Hg |
|---|---|---|---|
| 12:10 | 5 | .... | ... |
| 11 | 5 | .... | ... |
| 12 | 5 | .... | 104 |
| 13 | 5 | .... | 104 |
| 14 | 6 | .... | 104 |
| 15 | 5 | .... | 104 |
| 16 | 7 | .... | 104 |
| 17 | 5 | .... | 104 |
| 18 | 5 | .... | 104 |
| 19 | 6 | .... | 104 |
| 20 | 6 | 26.0 | 104 |
| 21 | 5 | 26.0 | 104 |
| 22 | 5 | 26.0 | 104 |
| 23 | 0 | 28.0 | 124 |
| 24 | 2 | 27.0 | 116 |
| 25 | 6 | 28.0 | 126 |
| 26 | 7 | 29.0 | 125 |
| 27 | 6 | 28.0 | 120 |
| 28 | 4 | 26.0 | 118 |
| 29 | 4 | 25.5 | 116 |
| 30 | 7 | 25.0 | 111 |
| 31 | 7 | 25.0 | 110 |
| 32 | 6 | 24.0 | 109 |
| 33 | .. | 23.5 | 109 |
| 34 | 6 | 23.5 | 108 |
| 35 | 8 | 23.0 | 106 |
| 36 | 7 | 22.5 | 104 |
| 37 | 8 | 22.5 | 104 |
| 38 | 7 | 23.0 | 105 |
| 39 | 8 | 22.5 | 104 |
| 40 | 3 | 22.5 | 104 |
| 41 | 12 | 22.0 | 104 |
| 42 | 8 | 21.5 | 105 |
| 43 | 8 | 21.0 | 104 |
| 44 | 10 | 21.0 | 104 |
| 45 | 9 | 21.0 | 106 |
| 46 | 10 | 21.0 | 106 |
| 47 | 6 | .... | 106 |
| 48 | 12 | .... | ... |
| 49 | 11 | .... | ... |

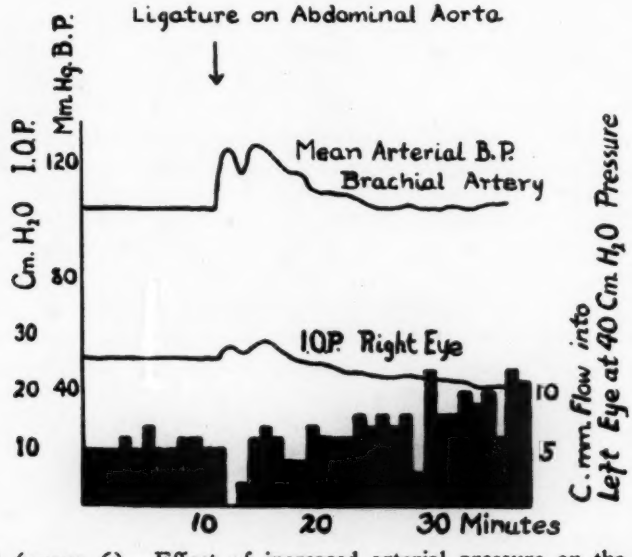


Chart 4 (exper. 6).—Effect of increased arterial pressure on the intra-ocular pressure, and on the rate of reabsorption of fluid from the anterior chamber.

### EFFECT OF LOCAL VASODILATATION

A third corollary to the hypothesis which we have outlined is that hyperemia of the iris and of the vessels connected with Schlemm's canal should cause an increased rate of absorption of fluid from the anterior chamber both at normal pressure and at the pressure level of the plateau. The low intra-ocular tension commonly found in cases of acute iritis and keratitis supports the truth of this contention. Similar

TABLE 7.—*Data in Experiment 7*

| Time, P.M. | Pressure, Cm. $H_2O$ | Flow, C.Mm. | |
|---|---|---|---|
| 4:26 | 43 | .. | |
| 27 | .. | 8 | |
| 28 | .. | 7 | |
| 29 | .. | 7 | |
| 30 | .. | 7 | |
| 31 | .. | 6 | |
| 32 | .. | 8 | |
| 33 | .. | 8 | |
| 34 | .. | 6 | |
| 35 | .. | 9 | |
| 36 | .. | 8 | Instilled 0.5% |
| 37 | .. | 7 | $NH_4OH$ |
| 38 | .. | 8 | |
| 39 | .. | 8 | |
| 40 | .. | 10 | |
| 41 | .. | 10 | |
| 42 | .. | 9 | |
| 43 | .. | 11 | |
| 44 | .. | 9 | |
| 45 | .. | 10 | |
| 46 | .. | 9 | |
| 47 | .. | 11 | |
| 48 | .. | 12 | |
| 49 | .. | 9 | |
| 50 | .. | No record | |
| 51 | .. | 11 | |
| 52 | .. | 12 | |
| 53 | .. | 13 | |
| 54 | .. | 13 | |
| 55 | .. | 15 | |
| 56 | .. | 13 | |
| 57 | .. | 13 | |
| 58 | .. | 16 | |
| 59 | .. | 16 | |
| 5:00 | .. | 18 | |
| 01 | .. | 9 | |
| 02 | .. | 13 | |
| 03 | .. | 20 | |
| 04 | .. | No record | |
| 05 | .. | 20 | |
| 06 | .. | 19 | |
| 07 | .. | 14 | |
| 08 | .. | 17 | |
| 09 | .. | 18 | |
| 10 | .. | 20 | |

conclusions are to be drawn from our own previously reported experiments on the effect of the injection of histamine into the anterior chamber, and also from the well known clinical effects of the instillation of concentrated solution of histamine into the conjunctival sac. Since in these instances marked effects on capillary permeability may be thought to complicate the interpretation, we have attempted to test the matter direcly.

EXPERIMENT 7 (Nov. 2, 1931).—A male dog weighing 8 Kg. was used. Anesthesia was produced with ¼ grain of morphine given hypodermically, followed

by 2.8 Gm. of sodium barbital intravenously. The blood pressure, recorded from the right femoral artery, remained constant throughout the experiment. A cannula was inserted into the right anterior chamber and connected, through the bubble manometer, with a reservoir of physiologic solution of sodium chloride raised to 43 cm. above the level of the eye. A steady flow of fluid into the eye was obtained. A few drops of dilute solution of ammonia were then instilled into the eye resulting in a marked conjunctival congestion, with hyperemia of the iris and miosis. The effect of this on the rate of reabsorption of fluid from the anterior chamber is seen in the following table and in chart 5.

*Comment.*—The increased rate of reabsorption of aqueous, increased venous pressure, increased arterial pressure and reflex vasodilatation all support the mechanical hypothesis of the method of reabsorption of the aqueous as outlined. Further study of the anatomic connections of Schlemm's canal and of the vasomotor control of the canal and its connections are required for the elaboration of this hypothesis. It must

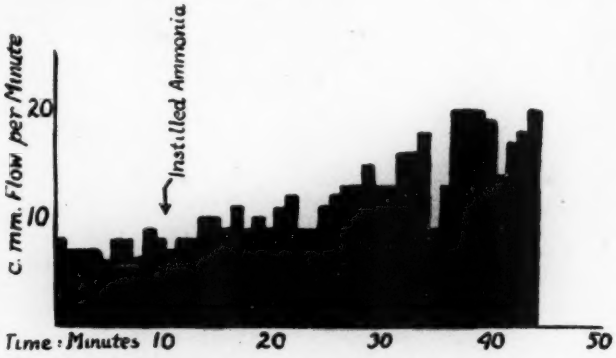


Chart 5 (exper. 7).—Effect of local vasodilatation on the rate of reabsorption of fluid from the anterior chamber.

also be noted that we have based this hypothesis on the conception that the vascular endothelium behaves, on the whole, as an inert semipermeable membrane, and that the force which drives fluid across this barrier is derived simply from the difference between the osmotic pressure on the two sides of the membrane and the difference between the hydrostatic pressure on the two sides of the membrane. That these forces, osmotic and hydrostatic, do, in fact, exert a dominant influence on the formation and reabsorption of the aqueous has been amply demonstrated. It is not inconceivable, however, that other forces may also play a part. Some inkling of the possible existence of such forces is to be found in the recent work on the transfer of fluid across membranes in virtue of differences in the surface electrical charges on the two sides of the membrane,[4] and in virtue of the difference in the hydrophilic swelling of the proteins on the two surfaces of the mem-

4. Freundlich, H.: Kapillarchemie, ed. 4, Leipzig, 1931, vol. 1.

branes.[5] A variety of such forces may contribute to the establishment of equilibrium between the blood and the aqueous. The present study has not taken these possible contributory factors into account for the simple reason that we have concerned ourselves solely with the effects of changes in hydrostatic pressure in the aqueous and blood vessels. So far as we can tell, we have disturbed the blood-aqueous equilibrium only in regard to these hydrostatic forces. The results which we have recorded can, therefore, be attributed to this disturbance of equilibrium irrespective of the complexity of forces involved in the normal maintenance of the equilibrium.

## CONCLUSIONS

1. The reabsorption of aqueous occurs almost exclusively from the anterior chamber; the tissues behind the iris absorb less than 1 per cent of the total flow.

2. The mechanism of reabsorption is normally capable of reabsorbing from five to ten times the normal rate of flow at pressures not more than from 3 to 10 mm. of mercury above the normal intra-ocular pressure.

3. Vasocongestion of the iris and pericorneal vessels due to venous stasis, to increased arterial pressure and to vasomotor reflex from corneal and conjunctival irritation, and from injection of histamine into the anterior chamber, increase the rate of reabsorption of the aqueous, and tend to lower the intra-ocular pressure, thus balancing and compensating for the increased rate of formation of aqueous which results from the same stimuli.

4. A simple hypothesis is formulated which accounts mechanically for these features of the mechanism of reabsorption of the aqueous.

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5. Wertheimer, E.: Arch. f. d. ges. Physiol. **201**:591, 1923; **208**:669, 1925.

# ABUSE OF THE THEORY OF FOCAL INFECTION

O. R. LOURIE, M.D.
BOSTON

Rosenow and Nickel's [1] experiments on elective affinity are of great interest to the scientist, independently of the practical value of the results of those investigations. The practitioner, however, is interested in the practical application of the theory of focal infection; he wants to know whether the elimination of the foci of infection serves its purpose; whether vaccine therapy is efficacious; whether it is proved that foci of infection are the cause of ocular diseases in most cases. Furthermore, he wants to know whether the results of the experiments are applicable to human beings, and can enlighten him in the treatment for ocular diseases.

## THE LAW OF LARGE NUMBERS

It is up to the bacteriologist to decide whether experiments on several hundred rabbits and guinea-pigs are sufficient to make definite conclusions on the subject of elective affinity. Haden's and Rosenow and Nickel's experiments show that there are rabbits that die after streptococcic inoculations; there are many that manifest elective affinity; there are many that do not react. Moreover, there are few that manifest ocular disease from streptococci of different affinity, and possibly there are some rabbits that may manifest ocular disease after being inoculated with sterile dextrose brain broth. The latter possibility can be inferred from the following sentence: [2] "Control intravenous and intratibial injections of like amounts of various batches of uninoculated glucose brain broth were made usually with negative results." If it were not so, the word "usually" would be supplanted with the word "always." The investigator depends on the kind of rabbits he chances to work with. If, for instance, the investigator happens to experiment on the kind of rabbits that do not manifest elective affinity, he would come to the conclusion that there is no such thing as elective affinity. On the contrary, if one chances to experiment on the kind of rabbits that manifest elective affinity, one would come to the opposite conclusion. The law of large numbers is especially important in such experiments, if one wants to avoid wrong conclusions based on insuffi-

1. Rosenow, E. C., and Nickel, A. C.: Elective Localization in Determining the Etiology of Chronic Uveitis, Am. J. Ophth. **15:1** (Jan.) 1932.
2. Rosenow and Nikel (footnote 1, p. 5.)

cient numbers. How can one be sure that, if thousands of rabbits were used, the results would be identical? It is known that Back [3] and Levy [4] could not obtain similar results in their experiments.

### HAS THE THEORY OF ELECTIVE AFFINITY A PRACTICAL VALUE?

Even if one assumes that Rosenow's theory of elective affinity is accepted by the whole scientific world, which in fact is not the case, still the question stands whether what is true in laboratory animals can be applied to human beings. In Rosenow's experiments the streptococcus has been isolated from a patient or rabbit with an ocular disease. This streptococcus, no matter where it comes from, frequently (not always) has manifested elective affinity for the eyes of rabbits. Perhaps the ocular disease produces some changes in the fluids of the whole body which cause the streptococcus or other micro-organisms to acquire elective affinity for the eye. This assumption may explain the results of inoculation of rabbits with the streptococcus from a human being with a diseased eye regardless of whether the streptococcus has lodged in the tonsils, teeth, cervix or elsewhere. But this does not mean that the streptococcus which has had no elective affinity for the human eye suddenly acquires it and causes the disease of the eye. It is obscure why and how the streptococcus acquires such an elective affinity in the human being. The latter carries the streptococcus in the teeth, tonsils or in other foci, and all of a sudden, without being inoculated with a special strain of a streptococcus with elective affinity for the eye, a disease of the eyes develops. Millions of human beings are carrying the streptococcus, but comparatively few develop diseases of the eye. Without elucidation of the causes of such transformation, the theory of elective affinity has no practical value.

### DO THE EXPERIMENTS IN THE LABORATORY REPRODUCE THE CONDITIONS THAT EXIST IN THE PATIENT?

Though Rosenow gives an affirmative answer to the question of whether experiments in the laboratory reproduce the conditions that exist in the patient, still it is permissible to doubt it. I have never seen, in my long practice, death from a disease of the eye except in a case of malignant tumor. A number of the experimental rabbits died. There must be a difference between an ocular disease in a human being and that produced in a poisoned animal. It seems to me that Rosenow's

3. Back, Heinrich: Klin. Monatsbl. f. Augenh. **78**:316, 1927.

4. Levy, J. M.; Steinbugler, W. F. C., and Pease, M. C., Jr. Investigations as to Frequency of Metastatic Eye Infections from Primary Dental Foci, J. A. M. A. **69**:194 (July 21) 1917.

experiments resemble more the cases of a general acute infection like relapsing fever, typhoid fever with involvement of the eyes, rather than the usual cases of ocular diseases which physicians meet most frequently in their practice. Between the acute or chronic diseases of the eye in a person that does not suffer from a generalized acute infection and the diseases of the eye produced artificially—there must be a great distance. One must not forget that the streptococcus has always been found in the diseased eyes of the inoculated animals but either never or only as an exception in cases of diseases of the eye in man. Verhoeff [5] spoke of the vessels loaded with streptococcus in the eyes of the inoculated animals. Wilmer [6] said: "The laboratory proof of pathogenic micro-organisms in the affected eyes suffering from dental sepsis has not been found." Benedict,[7] in answer to Goar's question whether organisms have been recovered from the human eye in cases of alleged iritis, from focal infection, said: "Yes, organisms appear in the aqueous in certain cases of severe iritis in humans." These facts show plainly that the blood stream of the inoculated animals contains the streptococcus, which is not the case or only an exceptional case in human beings. Intravenous injections and drilling of the bones with its unavoidable traumatization of the vessels do not reproduce the actual state of affairs that takes place in a patient.

### DOES THE THEORY OF ELECTIVE AFFINITY SOLVE THE WHOLE PROBLEM AND POINT THE WAY FOR SATISFACTORY TREATMENT?

Rosenow himself [8] expressed the following opinion concerning whether the theory of elective affinity solves the whole problem and points the way to satisfactory treatment:

> That removal or elimination, by non surgical means, of foci, so far as possible, with or without the use of autogenous vaccines, often results in spectacular cures and in prevention of attacks, and although of fundamental importance, is not, however, sufficient to solve the problem. More is required, such as specific treatment with an antiserum.

With two reservations, one can accept these conclusions: 1. The words "by non surgical means" should be omitted, as they are of little importance; the fact is that surgical means are just the means that are applied in the treatment for ocular diseases due to so-called foci of infection. 2. The words "often results in spectacular cures" should be

5. Verhoeff, cited by Gifford, S. R.: A Review of the Literature on the Etiology of Acute Iritis, Am. J. Ophth. **14**:108, 1931.

6. Wilmer, W. H.: The Relation of Teeth to Diseases of the Eye, Arch. Ophth. **4**: 466 (Oct.) 1930.

7. Benedict, cited by Rosenow and Nickel (footnote 1, p. 18).

8. Rosenow and Nickel (footnote 1, p. 16).

placed with "in some cases are followed by spectacular cures." As I pointed out in my previous paper,[9] the surgical elimination of the foci would be fruitless in the great majority of cases, even in accordance with the statistics of the advocates of the theory of focal infection, and "*post hoc* does not mean *propter hoc*."

With these two reservations one can accept Rosenow's conclusion that the problem has not been solved. I should like to believe that an antiserum will be efficacious. I remember Marmorek's antituberculosis serum, Gabritchevsky's antistreptococcic, antiscarlet fever serum and other serums which filled our hearts with hope, but in the course of time they have been discarded, so that even the names of the inventors have been lost for the younger generation. Billings was against therapeutic vaccination and for surgical removal of the foci of infection; others are for therapeutic vaccination; still others in their generosity are for therapeutic vaccination combined with operations, and finally Rosenow adds his hope for an antiserum. These facts show clearly that so far one possesses only fragments of knowledge on this subject. "*Noli nocere*" (do no harm) must be the first precept of a conscientious physician. Can one say that the mass operations performed for the removal of the foci of infection are harmless to the patients? Rosenow said:[10]

> Dismal failures have also been noted in some cases in which this latter procedure (removal of the foci and autogenous vaccine) had no apparent effect. In no cases we observed harmful effect.

The last assertion cannot be generalized, as most of the observers have seen most unfortunate results after all kinds of operations. In the November issue of the *Archives of Pediatrics,* Wall[11] described a case of pulmonary abscess following tonsillectomy and asserted that this complication occurs in 1:781 or 1:3,000 operations according to different authors. What about the complications like death, septicemia, hemorrhages, adhesions, nervous and vasomotor disturbances due to early sterilization of women, meningitis after sinus operations, harm done by numerous operations due to faulty diagnosis, etc.? Almost every specialist points to his field as the cause for trouble. With fragments of knowledge obtained in the experiments, which differ greatly from what is actually observed in human beings, how can one assert that there is a scientific foundation for elimination of the foci of infection? And how can one know where to look for them? After all kinds of operations there will still remain the mouth, with its mucous mem-

9. Lourie, O. R.: Focal Infection—an Etiologic Factor in Diseases of the Eye, Arch. Ophth. **5**:8 (June) 1931.

10. Rosenow and Nickel (footnote 1, p. 15).

11. Wall, Joseph S.: Lung Abscess Following Tonsillectomy, Arch. Pediat. **48**:687 (Nov.) 1931.

brane, and the nasopharynx, which are excellent seats for the streptococcus. There is such a thing as science, but there is also such a thing as common sense. While one reads Rosenow and Nickel's statistics on elective affinity based on experiments on several hundred laboratory animals, one should not forget Kaiser's and Cunningham's statistics on the results of tonsillectomies in many thousands of human beings. Kaiser also gives statistics [12] on the influence of tonsillectomies on the heart in rheumatic fever. It is of great interest to the oculist, because rheumatic iritis is not an uncommon complication in this disease.

### KAISER'S STATISTICS ON THE INFLUENCE OF TONSILLECTOMIES ON RHEUMATIC HEART DISEASE

Kaiser's statistics are impressive with their numbers: 450 diseased hearts in 20,000 tonsillectomized children; 817 diseased hearts in 28,000 children not operated on. In other words, there were 450 diseased hearts in 20,000 tonsillectomized children and 583 in 20,000 not operated on. The difference amounts then to 133 saved hearts for 20,000 operations, which means that to protect 133 children one has to victimize 19,867 children and remove 40,000 tonsils. I should not be surprised if I were told that the complications in the thousands of children on whom operation has been performed would average or exceed the number of saved hearts. But even the saving of these 133 children through the operation should be taken with reservations. The French have a good saying: *"comparaison n'est pas raison."* There are no two individuals alike. The children that have been compared were of different physical and mental make up, their resistance being different. Then again the infection could be of a different virulence. If one is to take into consideration this aspect of the question, the 133 children have possibly been saved through unknown factors rather than through tonsillectomy.

### THEORY OF FOCAL INFECTION TO BLAME FOR WHOLESALE REMOVAL OF TONSILS AND TEETH

As it is known, many observers consider the causal relationship between rheumatic fever and the tonsils beyond any doubt.

Is the cause of rheumatic fever known? This question is answered by Robey [13] as follows: "While we are waiting for the discovery of the specific organism and serum we must give more thought to the foci of infection"—that is to say, the cause of the disease is unknown. If the

12. Kaiser, Albert D.: Incidence of Rheumatism, Chorea and Heart Disease in Tonsillectomized Children: A Control Study, J. A. M. A. **89**:2243 (Dec. 31) 1927.

13. Robey, W. H.: Cardiology, Old and New, New England J. Med. **205**: 995 (Nov. 19) 1931.

pathogenesis of rheumatic fever is unknown, why then should one assume that the tonsils or the teeth are the primary seat of the unknown micro-organism? I have seen cases of rheumatic iritis in which the tonsils and the teeth had been removed long before the first attack of rheumatic fever. Even in the camp of the supporters of the theory, there is no unanimity. Robey,[13] for example, said:

> In most articles on rheumatic heart disease the tonsils are placed first in the list of causes; yet when various authors discuss the subject, they express grave doubt about the value of tonsillectomy, claiming that the operation does not prevent subsequent attacks of rheumatic fever.

The strict adherents of the theory of focal infection explain the recurrences in this way[13]: 1. The operation may be imperfectly performed and tonsillar remains be left which act as a focus in subsequent attacks. 2. If the tonsils have been allowed to remain through repeated attacks, they may succeed in establishing other foci of infection, even though eventually there has been a complete tonsillectomy.

This would mean that, if an attack of rheumatic fever occurred, the operation may prove futile because it could be incomplete, or new foci have already been established. The advocates of the theory do not know the exact time that is necessary for such foci to be established in different parts of the body, whether it takes a minute, an hour, a week or a year. Without this knowledge one is completely lost. It is clear that the advocates of the theory, if they are to be consistent, must insist on preventing rheumatic fever through a complete enucleation of healthy tonsils, because one cannot be sure that the tonsils are healthy, even though they appear to be so, and also because the removal of the tonsils during or after an attack of rheumatic fever may prove, and most frequently does prove, futile. In the words of Robey,[14] "rheumatic fever under two years of age has been authentically reported, but is very rare." Consequently, one must come to the mass removal of the children's tonsils which is going on in this country to the detriment of the population. As I pointed out in my previous paper,[9] one must also come to the wholesale removal of the teeth, if one accepts the assertions of the advocates of the theory. According to Dr. Peck of New Haven, one third of all operations in this country since 1924 were tonsillectomies. It becomes a serious social problem. The city council in Boston[15] demanded that the city hospital abolish the limit for the number of tonsillectomies performed every day, because the waiting list is so long that those registered in November can not be promised an operation before the following September.

If the immortal Voltaire had lived up to the present time, he certainly would have changed his definition of a physician from "a man

14. Robey (footnote 13, p. 993).
15. The Boston Herald, Nov. 24, 1931, front page.

that pours drugs of which he knows little into a body of which he knows less" to that of a man that removes tonsils of which he knows little from a body of which he knows less.

### REASONS FOR SPREAD OF THEORY OF FOCAL INFECTIONS

One of the most authoritative oculists in the country in a letter to me expressed the following opinion:

> The subject of focal infections is, of course, a very complicated one, and you are undoubtedly correct in criticizing the wholesale elimination of so-called sources of infection. This situation I think can practically be summarized as being due: (1) to ignorance; (2) unwillingness to take pains of a thorough medical examination; (3) response to the patient's wishing that something radical be done.

Other explanations can be offered. May I add three? 1. Some physicians believe in Billings' book as other people believe in the Bible. They are *"plus royalistes que le roi"* and can hardly be dissuaded. 2. Many others are attracted by the theory because it gives a ready remedy and solves their doubts, if only for a while. 3. Medical schools impress on the minds of students the indisputability of the theory.

Let me end with a quotation from Wall's [16] paper:

> Whether a fundamentalist, modernist, or evolutionist believing in the theory of vestigial remains, he should not look upon the presence of tonsils as an offense against the dictates of modern society, but rather upon their removal as an offense against Nature. It is not an uncommon experience for us to observe that the body retaliates for this insult in the destruction of her tissues by a process of replacement of tonsil-like lymphoid structures about the tonsil pillars and lower pharynx in an effort to restore that which has been ruthlessly taken from her.

It is hoped that science will solve these painful and perplexing problems. But true science never can be at odds with common sense.

### CONCLUSIONS

1. The beneficial results claimed by the supporters of the theory of focal infection are insignificant in comparison with the sacrifices involved due to operations.

2. The mass removal of tonsils is the result of insufficient knowledge of the causes of rheumatic and other infections. Insufficient knowledge, as a rule, should not be taken as an indication for an operation. One must not forget that practice of medicine is not experimentation in a laboratory.

3. The theory of elective affinity, as it stands, does not solve the problem of focal infection and can hardly be of help in the treatment for ocular diseases.

485 Commonwealth Avenue.

16. Wall (footnote 11, p. 701).

# LIGHTING WITHOUT GLARE

C. E. FERREE, PH.D.
AND
G. RAND, PH.D.
BALTIMORE

During the past several years we have designed a series of lighting units that are completely free from glare. One of these is a ward light for hospitals.

There has long been a need in hospitals for a lighting unit that combines the features of complete absence of glare and a satisfactory night-light attachment. The unit to be described in this paper was devised in response to a personal request from the director of one of our leading hospitals for a unit of this type. It has been in satisfactory use for more than a year in one of the new buildings of this hospital and has been installed in a second new building just completed for use.

## PRINCIPLES OF LIGHTING

It may be well to preface the description of the unit with a brief discussion of principles as applied to lighting by ceiling fixtures. Thus far ceiling fixtures have been of two types: (*a*) open bowls, opaque or translucent, installed pendant or inverted, and (*b*) closed bowls or other enclosing housings of translucent material. The situation with reference to these fixtures may be summarized briefly as follows. With an opaque reflector turned up (totally indirect lighting), the upper part of the room or other enclosure illuminated is too bright, especially when ceiling fixtures are used and high intensities of light are wanted on the working plane. To one lying in bed, the brighest surface in the room obviously should not be the ceiling. With an opaque reflector turned down (direct lighting), the upper part of the room is too dark and the lower part disproportionately bright, and the glare from the opening of the reflector is fatiguing and harmful to the eye. The glare from such a fixture is particularly annoying to one in a recumbent position. With a translucent reflector turned up (semi-indirect lighting), harmful glare results from the unit itself when the reflector is of too low density and from the ceiling when it is of high density. With a translucent reflector turned down (direct lighting with a translucent shade), the opening,

From the Research Laboratory of Physiological Optics, Wilmer Ophthalmological Institute, Johns Hopkins Medical School.

the sides of the reflector or both are of harmful brilliancy, the ratio of brilliancy of side and opening depending on the density of the reflector. With both the opaque and the translucent reflectors turned down, there is usually also harmful glare from the work because of the directed beam of light. In case an enclosing globe or housing of diffusing glass is used, a better distribution of light and surface brightness on walls and ceiling is obtained, but the glare from the source is cut down only in proportion as there is absorption of light by the enclosing glass. Because of this limitation, such units invariably have much too high a surface brilliancy.

In the practice of the lighting of interiors three means have been used, singly or in varying degrees of combination, for reducing glare from the source, namely, reflection, absorption and increase of the area of surface acting as the source. Clearly the first two are effective only in proportion as they are wasteful of light. The third has reached its highest development in flood-lighting and in the lighting of interiors with indirect lighting from floor and table stands, in which case the ceiling or some other large surface acts as a secondary source for illuminating the plane of work. It has never been made satisfactorily effective, however, with the use of any type of ceiling fixture.

It is obvious that in the lighting of interiors with ceiling fixtures, some type of glare-baffle is needed which will protect the eye from glare without harmful waste of light. With the demand for higher intensities of light, this need has become imperative. The use of higher intensities of light has also caused an increase of glare from the work. With a given character of working surface, this type of glare reaches its maximum with a directed beam of light and its minimum with a completely diffused light. In order to protect the eye from glare from the work, every lighting unit should, therefore, be provided with a diffusing member or some means of diffusing the light.

To give satisfactory results, a ceiling fixture should combine the following features: 1. It should provide a means for illuminating the ceiling and work with light in the ratio of intensities needed to give a comfortable distribution of brightness in the field of view. This can be obtained by a suitable combination of the features of direct and indirect lighting. 2. Means should be provided for shielding the eye from glare from that part of the fixture that directs the light to the working plane. 3. The beam directed to the working plane should be diffused.

All of the foregoing features are embodied in the unit to be described. There is provided with it also a night-light attachment which supplies enough light to the room for the inspection of the patient and the objects in the room. This light is in part reflected to the ceiling and from there is diffused to the working plane, and in part passes

through the diffusing member and glare-baffle across the bottom of the fixture, directly to the working plane. When the light is turned on, no surface or object in the room has a brightness high enough to be sensed through the closed lids. The room is softly and uniformly illuminated with well diffused light of an intensity that is not high enough to waken a sleeping patient. The two sources of light, general and twilight, are on separate circuits, and both can be operated from the corridor by an attendant or by the nurse in charge.

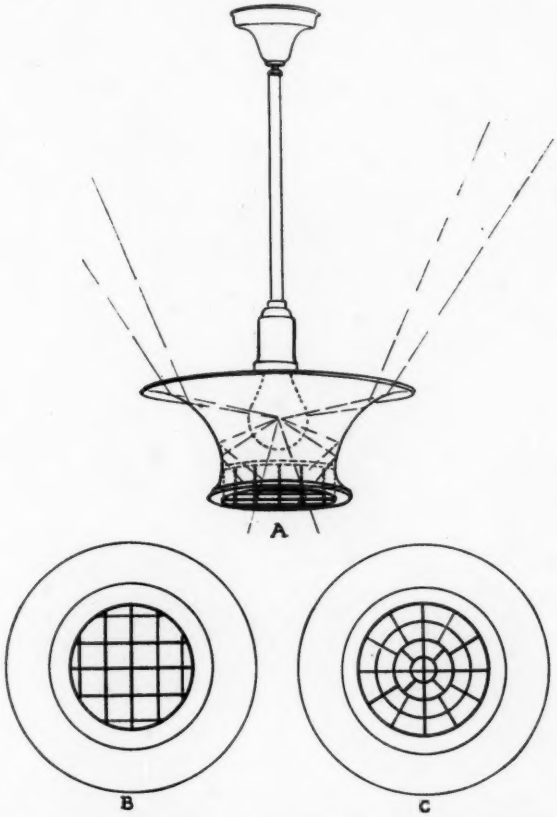


Fig. 1.—*A*, a perspective view of the unit in operative position; *B*, a bottom plan view of the glare-baffle, and *C*, a bottom plan view of another type of glare-baffle.

## THE LIGHTING UNIT

The unit is shown in the accompanying drawings. At *A* of figure 1 is a perspective view of the unit in operative position, and at *B*, a bottom plan view of the unit, showing in greater detail the glare-baffle. At the lower right at *C* is shown a bottom view of the unit with another type of glare-baffle. In figure 2 is shown a unit of a somewhat different shape and with still a different construction of baffle. This baffle

is represented in both bottom and side-bottom views at *B* and *C*, respectively. In figure 3, the unit is shown with the night-light attachment.

The construction will not be described in detail. At *A* of figure 1, it is seen that the upper part of the reflecting member is curved upwardly and outwardly to direct a portion of the light to the ceiling. The lower part of the reflector directs a part of the light from the lower hemisphere of the lamp to the opening in the bottom of the unit. The remainder of the light from the lower hemisphere of the lamp passes

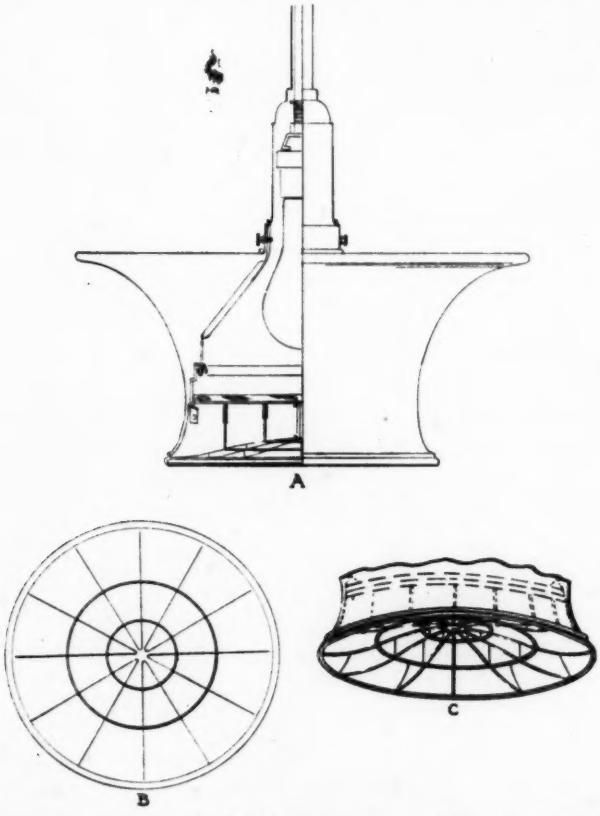


Fig. 2.—*A*, a second type of unit in operative position; *B*, a bottom plan view of the glare-baffle, and *C*, a side-bottom view of the glare-baffle.

directly from the lamp to this opening. This direct and reflected light is received by a plate of glassware, which extends across the unit at a suitable distance beneath the lamp. This plate, which has a high coefficient of both transmission and diffusion, serves to diffuse the light with a minimum loss by absorption. In the opening in the bottom of the unit, directly beneath the diffusing plate, is inserted the glare-baffle, represented by dotted lines in cross-section. This baffle, as is shown at *B*, has a cellular construction similar in appearance to an egg-crate. It

consists substantially of two series of vertical partitions extending across the opening at right angles to each other. The partitions extending in each direction are parallel. They are spaced apart by about 2 inches (5 cm.) and are approximately 2½ inches (6 cm.) wide. Through the downwardly extending passages formed by these partitions the greater portion of the light is allowed to pass to the plane of work, only such rays being intercepted as would produce glare in the eyes of those working or located in the various probable positions in the room. To one directly under the unit, or nearly so, glare is experienced only when the eyes are directed upward. In all other positions in the room the eye is protected in all directions. In order that the protection shall be com-

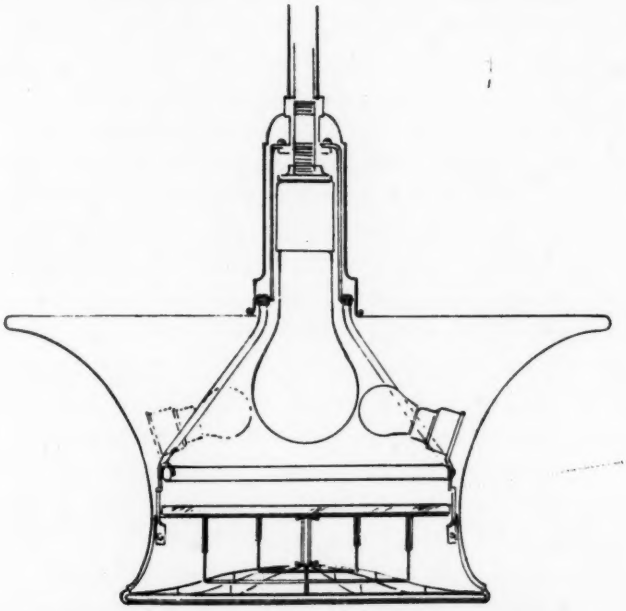

Fig. 3.—The unit with the night-light attachment.

plete, both surfaces of the partitions are painted a flat black. When the unit is installed in the proper position in the room and at the correct distance from the ceiling, the opening as seen by the patient in bed is either not luminous at all, or, if luminous, gives the effect of a dull luster or a pleasantly soft, silvery sheen of such low brightness as not to cause annoyance or discomfort to the most sensitive eye. For hospital purposes the unit also has the advantage that when used to give a low general illumination of the room the component of light passing through the opening at the bottom is strong enough to permit of the inspection of a chart or the reading of a thermometer or the doing of other things that require a comparatively high visibility.

At *C* of figure 1 the partitions are shown arranged in the form of concentric circles. Circular partitions, however, shield the eye completely only when the gaze is directed along the common radius of the several circles. In all other directions glare is experienced. In order to shield the eye in all directions, radial segments are inserted between the circular partitions at suitable places. While good results are obtained with this baffle, we have not found it as entirely satisfactory as the one shown at *B*.

A third type of baffle is shown at *B* and *C* of figure 2. This baffle affords as much protection to the eye as the one shown at *B* in figure 1 and gives a more artistic effect. The amount of light allowed to pass, however, is not so great. The reflector and baffle shown in figure 2 are the ones used in the hospital cited earlier in the paper. The partitions forming the baffle in this case are curved upward, so that the appearance is that of a bottom slightly convexed upward. Intersecting these partitions at slightly different vertical levels are two suitably spaced circular partitions. At *B* of figure 2 a bottom view is represented and at *C* a side-bottom view to show more clearly the shape and arrangement of the radial vanes used in this baffle. When illuminated and unilluminated, this baffle gives a pleasing effect. Because of its more artistic design throughout, the unit shown in figure 2 will in most cases doubtless be the one preferred.

In order further to protect the eye from glare, a downwardly extending canopy or glare shield is provided which encloses all the bright parts of the socket and the neck of the lamp. In the greater number of even the better lighting units in use at the present time these have a high brightness and are objectionable and disturbing to the eye. The canopy, which is attached to the lamp-socket, is shown in all the pictures given of the unit in operative position. It may be painted the same as the outer surface of the reflector, in the present unit light ivory, in which case it harmonizes with the reflector when the light is not turned on, or it may be given a darker shade to harmonize with the reflector when the light is turned on. In the latter case, a much better protection is afforded to the eye. In units so completely and uniformly glareless as these it is surprising to note what a small amount of brightness difference is required to become noticeable and even distractingly annoying to a sensitive eye.

In the hospital noted earlier in the paper, 40 watt mazda lamps have been found to give sufficient general illumination for ward lighting and enough light beneath the unit for the reading of thermometers, the inspection of charts and other work requiring a comparatively high visibility. The walls and ceiling of these wards are painted light ivory. Walls and ceilings of good reflecting power without gloss are a great aid in secur-

ing good lighting effects. The illumination given is soft, pleasing and comforting to the eye; the unit itself is glareless, and there is a well balanced distribution of light in the upper and lower halves of the room.

The night-light attachment is shown in figure 3. It is mounted inside the upwardly extending reflecting member, a short distance above the diffusing plate. It consists of a circular brass tube on which at diametrically opposite points are mounted two small frosted lamps (2 candle-power, 120 volt, form I, S-8, candelabra base). The filaments of these lamps are at approximately the same vertical level as the filament of the lamp that provides the general illumination. The feed wires for the lamp are threaded through the circular tube. The tube is held in position by suitable mounts depending from the main socket of the fixture. Two lamps were used for the following reasons: 1. If only one were used, the lamp employed for general illumination with its auxiliary fittings, which remain in position when the night-light is turned on, would cast troublesome shadows. 2. If more than two were used, the resultant intensity of illumination would be too high for a satisfactory night-light. Two lamps in the location described gave a shadowless, well distributed illumination of optimum intensity, or very nearly so.

While the unit in question is described as a hospital unit, the same glareless features that make it suitable for a hospital unit give it merit for other lighting situations. Without the night-light attachment, the unit serves in general an excellent purpose for commercial and home lighting, and in combination with the night-light attachment it has additional uses in the lighting of bedrooms and other rooms, halls, corridors, etc., in which a low as well as a high illumination may be needed.

The unit combines, it will be remembered, the features of diffused and glareless direct lighting with indirect lighting. The former provides for a strong downward component of diffused light without glare on desks, tables and other objects in the lower half of the room, and the latter gives a pleasing illumination to the upper half of the room and in addition an overlay of highly diffused and well distributed reflected light on objects in the lower part of the room. The purpose has been to secure in one unit the best features of both direct and indirect lighting in supplementary relationship. Neither type of lighting is complete in itself. To those who wish to combine welfare and comfort of the eye with a pleasing and artistic effect, the unit is recommended, and to the thousands of helpless sick who are suffering from harsh and improper lighting, the unit is dedicated.

In conclusion, a somewhat specialized use of the unit in combination with the night-light attachment may be noted, namely, the lighting of dark rooms. For the lighting of dark rooms the combination of the

features of low and high illumination with a complete absence of surfaces of high brightness in any part of the room satisfies a long-felt need. In fact, our own first use of the principles involved in the unit was for the illumination of the dark rooms of our laboratory. Their suitability for this purpose is obvious. We used at first a system made up of four direct units with a diffusing plate and a baffle across the bottom of each, located and suitably spaced at the corners of a square, and one indirect unit at the center of the square, the two types of unit being installed on separate circuits. The indirect unit was intended to give a low general illumination and the direct units the high light that was needed at times on objects in the working plane. A later thought, in conjunction with the designing of a hospital unit, was to combine both in the same housing and to provide two intensities of illumination. This combination has been found to be not only more convenient, but also more satisfactory from the standpoint of the lighting effects produced. In the unit as now constructed, a choice of the following conditions is provided: total absence of light for dark adaptation and other work that requires the complete exclusion of light; a low general illumination giving just the degree of visibility that is needed for the convenient use of the slit-lamp, the ophthalmoscope and the retinoscope and for other work in which complete exclusion of light is not required, together with a downward component of light to provide a comparatively small area of higher visibility which is often needed in work in the dark room, and a high illumination for use when arranging and setting up apparatus, when cleaning the room and for other utilities of various kinds. The provision of a means of illumination of this type not only removes many of the annoyances and inconveniences that are ordinarily associated with work in the dark room, but opens up new possibilities for the utilization of a standardized low illumination for the study of pathologic cases and for other purposes for which a low illumination is required.

# CHEMICAL COMPOSITION OF THE BLOOD AND SPINAL FLUID IN PRIMARY GLAUCOMA

## COMPARATIVE STUDIES

MARTIN COHEN, M.D.

JOHN A. KILLIAN, PH.D.

AND

LENA HALPERN, M.A.

NEW YORK

It is generally accepted that the intra-ocular and spinal fluids are a dialysate of the blood, and also that the vitreous filtrate of animals and the spinal fluid of man are of similar composition; therefore, a study of the composition of the human spinal fluid may provide information concerning the chemical composition of human intra-ocular fluid, which obviously is not available for analysis. Analysis of the blood of both man and animals shows great constancy of the organic and inorganic constituents. The increase of the intra-ocular fluid is the most important factor in the causation of the symptoms of primary glaucoma. We believe, with others, that this increase of intra-ocular pressure has a physicochemical basis, although this hypothesis has not as yet been proved.

With a consideration of these facts, we started our present study. For the past seven years we have been engaged in the physicochemical study of the intra-ocular fluid of normal animal eyes, and also the physicochemical relationship of the intra-ocular fluid to the blood and spinal fluid of animals. Following this preliminary study of the normal animal eye, we have, during the past year, investigated the blood and spinal fluid of patients with primary glaucoma. Fourteen patients affected with primary glaucoma were admitted, at different periods, to the wards of the Post-Graduate Hospital. Ten patients with ocular conditions who showed no clinical symptoms of glaucoma were also admitted. On admission (without breakfast), the patients were examined physically to rule out any general infections. Then specimens of blood and of spinal fluid were removed from each patient and sent to the laboratory. The patients remained in the hospital the entire day,

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This investigation was made possible by a grant from the Oliver Rea Fund.

Read before the Section of Ophthalmology, New York Academy of Medicine, Jan. 18, 1932.

TABLE 1.—*Primary Glaucoma*

| No. | Name | Age | Sex | Diagnosis | Duration | Vision | Tension (Schiötz) Average | Field | Disk | Treatment |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | R. B. ..... | 59 | F | Glaucoma simplex, R-L | 4 yrs., R-L | R = 20/70 L = 20/50 | R = 48 L = 45 | Glaucomatous, R-L | Moderate cupping, R-L | Trephining, R-L |
| 2 | J. S. ..... | 63 | M | Absolute glaucoma, R-L | 3 yrs., R-L | No light perception, R-L | R = 26 L = 21 | None, R-L | Cupping, atrophy, R-L | Trephining, R-L |
| 3 | R. H. ..... | 61 | F | Absolute glaucoma, R-L | 6 yrs., R-L | No light perception, R-L | R = 30 L = 30 | None, R-L | Cupping, atrophy, R-L | Trephining, R-L |
| 4 | H. K. ..... | 56 | M | Chronic inflammatory glaucoma, R-L | 1½ yrs., R-L | R = 20/30 L = 20/30 | R = 25 L = 30 | Slight nasal contraction, R-L | Cupping slight, L; none, R | Iridectomy, R-L |
| 5 | A. B. ..... | 63 | M | Glaucoma simplex, R-L | 4 months | R = 20/30 L = light perception | R = 30 L = 43 | Glaucomatous, R-L | Cupping, atrophy, R-L | Miotics |
| 6 | S. H. ..... | 42 | M | Glaucoma simplex, R-L | R, 2 yrs.; L, 3 yrs. | R = 20/70 L = light perception | R = 30 L = 18 | Glaucomatous, R-L | Cupping, R; atrophy, L | Trephining, L |
| 7 | E. S. ..... | 62 | M | Chronic inflammatory glaucoma, R | 3 yrs., R | R = 20/200 L = 20/20 | R = 28 L = 18 | Glaucomatous, R | Cupping, R; left, normal | Iridectomy, R |
| 8 | A. Y. ..... | 63 | M | Chronic inflammatory glaucoma, L | 1½ yrs., L | L = light perception | L = 18 | Glaucomatous, L | Cupping, L | Trephining, L |
| 9 | I. K. (Jap.) | 43 | M | Glaucoma simplex, R-L | 2 yrs. | L = fingers 6 inches R = 20/20 | R = 45 L = 45 | Glaucomatous, R-L | Cupping, R; atrophy, L | Medication |
| 10 | A. S. ..... | 55 | F | Glaucoma simplex, R-L | 10 yrs. | L = 20/20 R = 20/20 | R = 21 L = 35 | Glaucomatous, R-L | Cupping, R-L | Trephining, L |
| 11 | M. T. ..... | 48 | F | Glaucoma simplex, R | 2 yrs. | R = 2/200 L = 20/20 | R = 45 | Glaucomatous, R | Cupping, R | Trephining, R |
| 12 | L. D. (col.) | 52 | F | Glaucoma simplex, R-L | 3 yrs. | L = P.L. R = 20/50 | R = 35 L = 35 | Glaucomatous, R-L | Cupping, atrophy, R-L | Trephining, R-L |
| 13 | C. S. ..... | 65 | M | Glaucoma simplex, R-L | 6 yrs. | R = 5/200 L = P.L. | R = 13 L = 43 | Glaucomatous, R-L | Cupping, atrophy, R-L | Trephining, L |
| 14 | A. S. ..... | 45 | F | Glaucoma simplex, R-L | 2 months | R = 20/30 L = 20/30 | R = 60 L = 60 | Glaucomatous, R-L | Cupping, atrophy, R-L | Trephining, L |

and left without showing the slightest ill effects. The patients with glaucoma were of three types: Nine had glaucoma simplex, two absolute glaucoma, and three primary inflammatory glaucoma. The disease had existed in these patients for from two months to ten years; the average time was two years. There were eight male and six female patients, all between 45 and 65 years of age. In twelve, both eyes were involved; in the other two, only one eye was affected. The treatment of these patients with glaucoma previous to admission had consisted in trephining in nine cases, iridectomies in three and the use of miotics in two. The cases presented the characteristic clinical symptoms of primary glaucoma. In all, the intra-ocular pressure, measured with the Schiötz tonometer, was abnormally high, and the visual fields were typical of glaucoma; in the majority of the patients, the fundus showed cupping of the disk with atrophy (table 1).

TABLE 2.—*Controls*

| Number | Name | Age | Sex | Diagnosis |
|---|---|---|---|---|
| 1 | L. D. | 37 | Female | Dacryocystitis |
| 2 | L. O. | 70 | Female | Cataract |
| 3 | M. S. | 45 | Female | Trachoma |
| 4 | A. R. | 31 | Female | Cataract |
| 5 | M. T. | 54 | Male | Cataract |
| 6 | C. A. | 52 | Female | Cataract |
| 7 | A. S. | 50 | Male | Iritis (traumatic) |
| 8 | J. B. | 39 | Male | Irido cyclitis |
| 9 | E. K. | 18 | Male | Cataract |
| 10 | K. H. | 48 | Male | Pterygium |

The ten nonglaucomatous patients consisted of five with cataracts, two with iritis, one with trachoma, one with dacryocystitis and one with a pterygium. The ages of these patients corresponded to the ages of the patients with glaucoma (table 2).

When the chemical analyses of the blood and the spinal fluid obtained from these two groups of patients were completed, the results were compared. The chemical analysis of this report will be presented later in the article.

## COMMENT

It is, of course, possible to have the chemical composition of the intra-ocular fluid in patients with glaucoma not exactly the same as that of the spinal fluid, on account of local changes in the ocular media, which are not necessarily reflected in the spinal fluid.

A comparative study of the cases in this report indicates that while individual analyses are variable, the average chemical analyses of the blood and the spinal fluid in glaucomatous and nonglaucomatous patients, or controls, are similar.

REVIEW OF THE LITERATURE

A search of the literature for analyses of the blood of glaucomatous patients yields extremely meager, incomplete and confusing results. Owing to the interest aroused by the various investigations [1] showing swelling of the vitreous in alkaline mediums, attention has been concentrated on determinations of the $p_H$ of the body fluids in glaucoma.[2] The general conclusion is, however, that the reaction of the blood is of no primary importance here and that further studies along the same lines are fruitless. There have been various reports on a possible hypercholesteremia in the disease, and, although the evidence is not unanimous, the conclusion seems justified that cholesterol plays no significant rôle.[3] Petersen and Levinson, by averaging the results in fourteen patients with glaucoma, found among other things that the globulin was slightly low, but that the potassium, calcium, potassium-calcium ratio, sugar and capillary permeability were close to normal. Each case, however, presented definite evidence of alteration referable to the vascular system. Schmidt was unable to find any disturbance of capillary permeability by the Marx drinking method, which, however, shows wide variations in the normal reaction.[4] Rowe [5] found no significant endocrine factor in glaucoma, but of forty-eight cases studied by Passow,[6] 83 per cent showed an increase in basal metabolism; of fifty-one cases, 84 per cent showed a hyperiodemia, and of thirty-three cases, 48 per cent showed an increase in vasoconstricting substance (epinephrinemia), while glycemia, an increased red blood cell and mononuclear count and decreased choline were often observed. Furthermore, the calcium was slightly high and the potassium low. In a clinical study by Gouterman,[7] it was pointed out that treatment with calcium chloride or parathyroid and ultraviolet radiation or viosterol was efficacious in reducing intra-ocular pressure, but no direct evidence was given for a calcium deficiency in glaucoma.

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1. Redslob, E., and Reiss, P.: Ann. d'ocul. **166**:1, 1929. Duke-Elder, W. S.: Tr. Ophth. Soc. U. Kingdom **49**:83, 1929.

2. Gala, A.: Brit. J. Ophth. **9**:516, 1925. Schmerl, E.: Ann. d'ocul. **165**:565, 1928.

3. Musselevitch, A. L.: Russk. arch. oft. **7**:529, 1930. Passow, A.: The Significance of Lipoids in Primary Glaucoma, Arch. Ophth. **4**:781 (Nov.) 1930. Salvati: Ann. d'ocul. **163**:363, 1926. Petersen, W. F., and Levinson, S. A.: The Skin Reactions, Blood Chemistry and Physical Status of "Normal" Men and of Clinical Patients: V. Patients with Glaucoma and Their Vascular Reactions, Arch. Path. **9**:282 (Jan.) 1930.

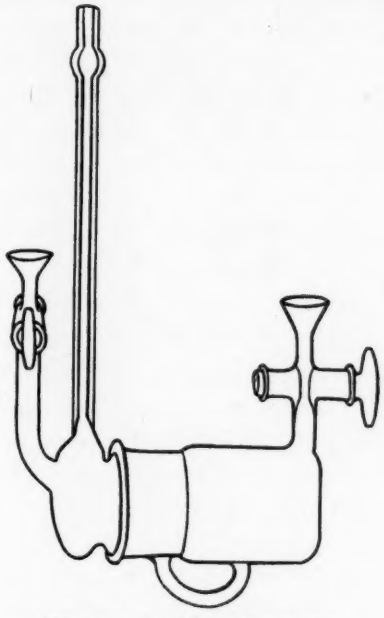
4. Schmidt, K.: Arch. f. Augenh. **98**:569, 1928.

5. Rowe, A. W.: Endocrine Studies: XXIV. The Etiology of Glaucoma, Arch. Ophth. **3**:403 (April) 1930.

6. Passow, A.: Arch. f. Augenh. **103**:111, 1930.

7. Gouterman, J. I.: The Rôle of Calcium in Essential Glaucoma, Arch. Ophth. **2**:670 (Dec.) 1929.

The possibility that calcium is somehow concerned with the production of an increased intra-ocular pressure suggests itself strongly because of its ability to decrease the excitability of the cervical sympathetic nerve and the permeability of the capillaries.[8] It is not the total calcium but the diffusible, or perhaps only the ionic, calcium that is concerned with this reaction. The determination of ionic calcium is, as yet, out of the realm of practical laboratory procedure, while the analysis of serum for diffusible and nondiffusible calcium in spinal fluid is of more immediate interest, since the factor of capillary perme-



Vitreous swelling apparatus.

ability is also involved in the results. Although differing in actual concentrations, the spinal fluid is believed to be closely analogous to the intra-ocular fluids in concentration and method of formation.

It is well known that not only the $p_H$ but also the salt content influences the degree of swelling of lyophilic gels. Furthermore, certain ions exert a specific favorable or inhibitory effect on swelling, apart from the reaction or concentration of the medium. Since the swelling of the vitreous is probably an essential factor in glaucoma, it is desirable to know whether it is accompanied by a change in concentration of any of the ions normally occurring in blood, such as calcium, sodium or chlorides. For a better understanding of the condition of the calcium it is advisable to investigate the protein content of the blood

8. Kikai, K.: Arch. f. Augenh. **104**:134, 1931.

as well, since a normal calcium with a low protein count, especially a low globulin, indicates a high concentration of diffusible calcium which might not otherwise have been appreciated.

Although Duke-Elder [9] stated that a few investigators who have examined the blood of glaucomatous patients are unanimous in finding a decrease in salt content, we find this conclusion to be partly inaccurate owing to an error in the reading of the article of Ascher.[10] The latter stated that the sodium chloride in the aqueous of glaucomatous patients is as often normal as it is high or low, but the high figures are higher than in any other disease of the eye. In blood and serum, the sodium chloride content was within normal limits in glaucoma and gave no uniform results when compared with the salt content of the aqueous. According to Hertel,[11] the molecular concentration (by cryoscopy) and the proteins of the serum were at the lower normal. Hertel and Citron [12] and Pletnewa [13] concluded that on the average the osmotic pressure of the blood of persons suffering from glaucoma was less than that of healthy persons. Alexandre [14] reported that the viscosity of glaucomatous blood was lower than normal.

## OBSERVATIONS

We wish to report the results of a series of determinations in fourteen hospitalized patients with primary glaucoma and in ten controls with disturbances other than glaucoma on the eye ward of this hospital. The first series consists of eight men and seven women, ranging in age from 41 to 64 years, the average age being 55. The second series consists of six males and four females, ranging in age from 14 to 65 years, the average age being 46. The analyses presented were done immediately on the specimens of blood and spinal fluid withdrawn under fasting conditions (before breakfast). The methods employed were those that have been tested and used with satisfaction in the biochemistry laboratory of the New York Post-Graduate Hospital. The spinal fluid protein was determined by the procedure of Denis and Ayer; [15] the plasma proteins, by the method of Wu.[16] The sugar of both the blood and the spinal fluid was estimated by the method of Shaffer and

9. Duke-Elder, W. S.: Nature of the Vitreous Body, Brit. J. Ophth. (monog. supp.) **14**:609, 1930.

10. Ascher, K. W.: Arch. f. Ophth. **107**:247, 1922.

11. Hertel, E.: Arch. f. Ophth. **90**:209, 1915.

12. Hertel and Citron: Klin. Monatsbl. f. Augenh. **51**:351, 1913.

13. Pletnewa: Russ. ophth. J. **2**:7, 1923.

14. Alexandre: Thèse de Paris, 1912.

15. Denis, W., and Ayer, J. B.: A Method for the Quantitative Determination of Protein in Cerebrospinal Fluid, Arch. Int. Med. **26**:436 (Oct.) 1920.

16. Wu, H.: J. Biol. Chem. **51**:33, 1922.

Hartmann;[17] the chlorides, by the Van Slyke method,[18] and sodium, by the technic of Kramer and Gittleman.[19] The Clark-Collip modification of the Kramer-Tisdall[20] method was used for the determination of calcium, 4 cc. of spinal fluid being used in most cases. We used the Greenberg and Gunther modification of the Fiske and Subbarow[21] method for the determination of inorganic phosphates. Occasionally low results were obtained, which, however, were repeated by the direct Fiske-Subbarow method.[22] In the analyses of the data, we calculated the arithmetical averages for each series as well as the average deviation. The latter was obtained by adding up the deviation of each determination from the average, without regard for the algebraic sign, and dividing by the number of determinations. We considered the average for glaucomatous and nonglaucomatous patients to lie within the same range if the difference between them was not larger than the larger average deviation for that series. This can be more clearly understood when the individual results are examined.

Although Denis and Ayer[15] gave from 35 to 100 mg. of protein per hundred cubic centimeters as the normal range for the cerebrospinal fluid, our experience in this laboratory has shown that considerably lower figures are normally obtained. In this investigation for the nonglaucomatous series, the figures lie between 14 and 51.3 mg., with the average at 22.6 mg. and an average deviation of 5.2 mg. In the glaucoma series, the figures lie between 9.7 and 48.7 mg., averaging 24.9 mg., with an average deviation of 9.1 mg. The difference between the averages lies well within the range allowed by the larger average deviation (9.1 mg.); therefore, we may consider that there is no variation of spinal fluid protein from that in the control series.

Examination of table 3 shows that the averages for the fibrin, albumin, globulin and albumin-globulin ratio of glaucomatous blood do not differ from those for nonglaucomatous blood by more than the average deviation in each case, but show excellent agreement. The reports in the literature as to the normal range for plasma proteins as determined by the Wu method have been varied, but the results given here agree well with our experience in this laboratory. The conclusion seems justified that there is no disturbance in protein concentration or equilibrium in glaucoma.

With the Hartmann-Shaffer method of analysis, the normal range for blood sugar is from 70 to 110 mg. per hundred cubic centimeters. With

17. Shaffer, P. A., and Hartmann, A. F.: J. Biol. Chem. **45**:365, 1921.
18. Van Slyke, D. D.: J. Biol. Chem. **58**:523, 1923.
19. Kramer, B., and Gittleman, I.: J. Biol. Chem. **62**:353, 1924.
20. Clark, E. P., and Collip, J. B.: J. Biol. Chem. **63**:461, 1925.
21. Greenberg, D. M., and Gunther, L.: J. Biol. Chem. **82**:551, 1929.
22. Fiske, C. H., and Subbarow, Y.: J. Biol. Chem. **66**:375, 1925.

the exception of two cases (S. R. and K. H.), the analyses presented for the nonglaucomatous series lie within this range. The upper limit of the glaucomatous series was slightly higher than usual (114 mg.), but the averages for the two series compare closely. From 50 to 75 mg. per hundred cubic centimeters has been given as the normal limit for spinal fluid sugar by Fremont-Smith and Dailey.[23] This, however, was obtained by the Folin-Wu method of analysis. Killian, Osnato, Garcia and Mattice[24] have reported a study of spinal fluid sugar by the Hartmann-Shaffer method in epilepsy, in which the variations noted,

TABLE 3.—*Analysis of Data for Proteins*

| Fluid | Constituent | Number of Cases | Type of Case | Minimum | Maximum | Average |
|---|---|---|---|---|---|---|
| Spinal fluid | Total protein, mg. per 100 cc. | 10 | Control | 14.0 | 41.3 | 22.6 ± 5.2 |
| | | 14 | Glaucoma | 9.7 | 48.7 | 24.9 ± 9.1 |
| Plasma.... | Fibrin, per cent..... | 10 | Control | 0.29 | 0.58 | 0.40 ± 0.06 |
| | | 14 | Glaucoma | 0.25 | 0.51 | 0.39 ± 0.05 |
| Serum..... | Albumin, per cent..... | 10 | Control | 4.6 | 5.7 | 5.0 ± 0.4 |
| | | 14 | Glaucoma | 4.2 | 5.8 | 5.2 ± 0.4 |
| | Globulin, per cent..... | 10 | Control | 2.1 | 3.0 | 2.6 ± 0.3 |
| | | 14 | Glaucoma | 2.1 | 3.7 | 2.8 ± 0.4 |
| | Ratio—albumin to globulin | 10 | Control | 1.58 | 2.68 | 2.00 ± 0.27 |
| | | 14 | Glaucoma | 1.44 | 2.64 | 1.88 ± 0.20 |

TABLE 4.—*Analysis of Data for Sugar*

| Fluid | Number of Cases | Type of Case | Minimum | Maximum | Average |
|---|---|---|---|---|---|
| Spinal fluid..... | 6 | Control | 60 | 70 | 66 ± 3 |
| | 13 | Glaucoma | 44 | 81 | 63 ± 9 |
| Blood..... | 6 | Control | 76 | 110 | 94 ± 11 |
| | 13 | Glaucoma | 76 | 114 | 96 ± 11 |
| Ratio—spinal fluid to blood..... | | Control | 0.58 | 0.78 | 0.69 ± 0.04 |
| | | Glaucoma | 0.53 | 0.85 | 0.66 ± 0.08 |

with two exceptions, were from 48 to 82 mg. per hundred cubic centimeters, with an average of 74 per cent of that of blood. These figures are in better agreement with our results than those reported by Goodwin and Shelley,[25] who gave the normal spinal fluid sugar as from 45 to 65 per cent of that in blood, averaging 53 per cent. Our values for the controls, with the two exceptions noted, lie between 60 and 70 mg., averaging 66 mg., with an average deviation of ± 3 mg. The average spinal fluid-blood sugar ratio was 69.9 per cent, with an average devi-

23. Fremont-Smith, F., and Dailey, M. E.: Human Cerebrospinal Fluid, New York, Paul B. Hoeber, Inc., 1926, p. 104.

24. Killian, Osnato, Garcia and Mattice: Brain **50**:581, 1927.

25. Goodwin, G. M., and Shelley, H. J.: The Sugar Content of the Cerebrospinal Fluid and Its Relation to the Blood Sugar, Arch. Int. Med. **35**:242 (Feb.) 1925.

ation of ± 4.9. As can be seen from the table, the results for the patients with glaucoma are in good agreement with those for the control series.

A question might be raised as to the advisability of including in the series patients with pterygium and cataract, whose blood and spinal fluid sugars were outside the normal limits. Since the rest of the analyses in each case did not exceed the range of the rest of the cases, except for the spinal fluid chloride of S. R., which was slightly low, we deemed it advisable to include them in the nonglaucomatous series.

Reference to table 5 will show that the chlorides of blood and spinal fluid and their ratios are closely comparable for the patients with

TABLE 5.—*Analysis of Data for Chlorides*

| Fluid | Number of Cases | Type of Case | Minimum | Maximum | Average |
|---|---|---|---|---|---|
| Spinal fluid, mg. per 100 cc.... | 10 | Control | 721 | 753 | 737 ± 9 |
| | 14 | Glaucoma | 706 | 764 | 742 ± 14 |
| Blood, mg. per 100 cc..... | 10 | Control | 440 | 483 | 462 ± 14 |
| | 14 | Glaucoma | 440 | 492 | 469 ± 11 |
| Ratio—spinal fluid to blood.. | 10 | Control | 1.50 | 1.68 | 1.59 ± 0.04 |
| | 14 | Glaucoma | 1.49 | 1.68 | 1.58 ± 0.06 |

TABLE 6.—*Analysis of Data for Calcium*

| Fluid | Number of Cases | Type of Case | Minimum | Maximum | Average |
|---|---|---|---|---|---|
| Spinal fluid, mg. per 100 cc.... | 10 | Control | 4.6 | 5.2 | 5.0 ± 0.2 |
| | 14 | Glaucoma | 4.6 | 5.6 | 5.1 ± 0.2 |
| Serum, mg. per 100 cc..... | 10 | Control | 9.7 | 11.4 | 10.4 ± 0.4 |
| | 13 | Glaucoma | 9.7 | 12.3 | 10.8 ± 0.6 |
| Ratio—spinal fluid to serum.. | 10 | Control | 0.43 | 0.53 | 0.47 ± 0.02 |
| | 13 | Glaucoma | 0.42 | 0.54 | 0.47 ± 0.02 |

glaucoma and the controls. The accepted normal limits for blood are from 450 to 500 mg., and for spinal fluid, from 700 to 750 mg. With a few slight variations, our results fall within these extremes.

We were not able to find any disturbance of calcium metabolism and diffusibility in glaucoma. The limitations to the possibility of estimating the diffusibility of calcium by measuring its concentration in the spinal fluid must of course be borne in mind.[26] Merritt and Bauer [27] considered from 4.5 to 5.5 mg. per hundred cubic centimeters as normal for spinal fluid calcium. From 9.6 to 12 mg. is considered normal for serum, although the figures reported by the latter observers are lower. It is seen that, except for one case with a slightly high serum calcium (S. K.) and two cases with a slightly high spinal fluid calcium (J. S. and R. B.), our series lies well within the normal range.

Although the glaucomatous and nonglaucomatous cases compare closely when the averages for the inorganic phosphorus of spinal fluid

26. Greenberg, I. M.: Proc. Soc. Exper. Biol. & Med. **27**:514, 1930.
27. Merritt, H. A., and Bauer, W.: J. Biol. Chem. **90**:215, 1931.

and of serum are considered, the ratio between them appears to be somewhat higher in glaucoma. When the average deviation is considered, the variation is very small, but it nevertheless is present in this series. Cohen [28] found an average of 3.3 mg. of inorganic phosphorus for blood and 1.6 mg. for spinal fluid in six normal persons, the average for the spinal fluid forming 48 per cent of that of the blood plasma. These figures agree closely with the results reported by us.

The sodium of both blood and spinal fluid is usually given as from 300 to 320 mg. per hundred cubic centimeters. Our experience has

TABLE 7.—*Analysis of Data for Inorganic Phosphorus*

| Fluid | Number of Cases | Type of Case | Minimum | Maximum | Average |
|---|---|---|---|---|---|
| Spinal fluid, mg. per 100 cc.... | 10 | Control | 1.0 | 1.7 | 1.4 ± 0.2 |
| | 13 | Glaucoma | 1.2 | 2.0 | 1.6 ± 0.2 |
| Serum, mg. per 100 cc..... | 10 | Control | 2.2 | 4.5 | 3.3 ± 0.5 |
| | 13 | Glaucoma | 2.3 | 4.0 | 3.2 ± 0.4 |
| Ratio—spinal fluid to serum.. | 10 | Control | 0.33 | 0.50 | 0.42 ± 0.04 |
| | 12 | Glaucoma | 0.38 | 0.70 | 0.51 ± 0.06 |

TABLE 8.—*Analysis of Data for Sodium*

| Fluid | Number of Cases | Type of Case | Minimum | Maximum | Average |
|---|---|---|---|---|---|
| Spinal fluid..... | 10 | Control | 318 | 347 | 333 ± 8 |
| | 10 | Glaucoma | 308 | 332 | 324 ± 6 |
| Serum..... | 9 | Control | 305 | 337 | 322 ± 9 |
| | 10 | Glaucoma | 292 | 348 | 330 ± 15 |
| Ratio—spinal fluid to serum.. | 9 | Control | 0.98 | 1.09 | 1.03 ± 0.02 |
| | 9 | Glaucoma | 0.97 | 1.05 | 1.03 ± 0.02 |

yielded figures that are rather higher, and with the exception of one case of cataract, which shows a high serum sodium, and one case of glaucoma, which shows a low serum sodium, the cases of glaucoma show no abnormality of sodium concentration in the serum. The control series shows a slightly higher average for spinal fluid sodium than the glaucoma series, but the ratios compare easily within the range allowed by the average deviation.

## CONCLUSIONS

We may conclude, then, from the results given, that there is no disturbance in the plasma and spinal fluid proteins, the blood and spinal fluid sugar or chlorides, the serum and spinal fluid calcium and the inorganic phosphorus and sodium in chronic primary glaucoma. There may be a slightly high spinal fluid-serum inorganic phosphorus ratio. Our results show no striking difference between the blood and the spinal fluid of patients with glaucoma and those of patients suffering from other ocular disturbances.

28. Cohen, N.: Quart. J. Med. **17**:289, 1923.

## ABSTRACT OF DISCUSSION

DR. ARTHUR M. YUDKIN: We have found that the blood from patients afflicted with glaucoma, particularly glaucoma simplex, has in the main the same chemical ingredients as normal blood. This observation is in accord with that presented by Dr. Cohen, but differs considerably from that published in the foreign literature.

As for the instrument that was presented, I believe that one criticism can be made. The use of oil in the calibration section of the instrument is not very reliable, for it seems to me that it might interfere with the correct reading of the instrument. I believe that the results would be more accurate if mercury were used instead of oil.

DR. ERVIN A. TUSAK: I do not think that we should assume that any intra-ocular humor is analogous with the cerebrospinal fluid. We know that Treacher-Collins claimed that, partly or wholly, the ciliary glands secrete the aqueous. On the other hand, anybody who is vaguely familiar with colloid chemistry will know that there is quite a difference between the almost semicoagulated gel, the vitreous, if I may use that incorrect but plastic expression, and the cerebrospinal fluid, which is liquid in its aggregate condition. Furthermore, there is a certain histologic structure to the vitreous, and no histologic structure to the aqueous or cerebrospinal liquid. There is an added physicochemical difficulty that in all these fine physicochemical reactions we are dealing with equilibria through existing but entirely unexplored membranes which were not studied by the physiologist, morphologist or physical chemist. Furthermore, there are other things that cannot be circumvented, and one of these is that the blood is the collecting stream of all secretions of the human body, well buffered for any debalancing influence, such as, for instance, changes of $p_H$. The intra-ocular physiochemical changes influence the blood so slightly that parallel changes in the blood can hardly be noted with our present technic.

DR. MARTIN COHEN: I have nothing to add except to say that we are considering in these cases not the vitreous as a whole, but the vitreous filtrate compared with the spinal fluid, and I think that we can make a fair comparison. We simply present these analyses for your consideration.

DR. JOHN A. KILLIAN: With reference to the suggestion that we adopt a mercury manometer to measure the change in pressure due to a change in volume of the vitreous gel rather than an oil manometer, I wish to state that the use of a mercury manometer would necessitate attaching the buret to the base of the chamber, and furthermore, this would introduce many technical difficulties in filling the chamber and adjusting the apparatus. Moreover, the changes in height of the mercury column would be very small in comparison with the changes in volume of the vitreous gel. We have adopted the use of mineral oil because of the ease of layering it over the contents of the chamber and also because its use obviates a source of error due to evaporation during the long incubations necessary in these experiments.

With reference to Dr. Tusak's discussion, I wish to make it clear that we do not subscribe to the theory that the humors of the eyes are derived from the blood plasma by any process of filtration or dialysis similar to the assumed derivation of spinal fluid from blood plasma. The only reason for considering the composition of spinal fluid in these studies on glaucoma was because it gave us an index of diffusion of these substances into what are assumed to be transudates.

# SIMPLE METHOD FOR CALCULATING DIOPTRIC POWER

JOSEPH I. PASCAL, M.D.
BOSTON

The methods most in use for calculating the refracting power of a surface or a lens generally involve the use of formulas embodying several fractions. This is necessitated by the fact that the relation between dioptric power and focal length and that between curvature and radius are mathematically reciprocal relations, that is, inverse relations. The longer the focal length, the less is the power; similarly, the longer the radius, the less the curvature. Memorizing formulas expressed in fractions and carrying out calculations by means of fractions are processes distasteful to most ophthalmologists, especially if they have not had much mathematical training.

A method that I have used for many years in teaching this phase of physiologic optics partly eliminates these difficulties. It provides an easily memorized formula and involves a knowledge of only the elements of arithmetic and algebra for calculating dioptric powers. For this purpose I have introduced the use of a *unit of curvature* similar to the unit of power. It will be recalled that the early method of numbering lens power by the inch system involved the use of cumbersome fractions because the power, as the reciprocal of the focal length in inches, had to be expressed as a common fraction. The introduction of the diopter as a unit of power, 1 diopter representing the focusing power corresponding to a focal length of 1 meter, eliminated the use of fractions and greatly simplified the procedure of combining lenses in examining and in prescribing.

The introduction of a similar unit of curvature likewise simplifies the calculations of power, as will be readily seen. I have adopted the term "metrec" [1] for a unit of curvature corresponding to a radius of 1 meter (40 inches, approximately). A radius of 50 cm. gives a curvature of 2 metrecs; 25 cm., 4 metrecs; 10 cm., 10 metrecs, etc. This is exactly the same as the relation of focal length to diopters. A focal length of 1 meter gives a power of 1 diopter; 50 cm., 2 diopters; 25 cm., 4 diopters; 10 cm., 10 diopters, etc. When the radius is given in milli-

1. This word is the simplest contraction of "meter-curvature," the c at the end reminding one, if that is necessary, that it is a unit of curvature. The word sounds strange at first but becomes perfectly assimilated after one uses it in a few calculations.

meters, as for example in calculating the surface powers of the eye, the radius is divided by 1,000 to get metrecs of curvature, just as when the focal length is given in millimeters this is divided by 1,000 to get diopters of power. The anterior surface of the cornea, for example, has a radius of about 8 mm.; its curvature corresponding to this radius is therefore 125 metrecs (1,000 divided by 8).

The power of a surface separating two media can be expressed either by the reciprocal of its anterior focal length or by the reciprocal of its posterior focal length. The usual method is to express it by the reciprocal of its anterior focal length. This, which may be called its anterior power, represents the power of the surface to make incident, divergent or convergent light parallel after refraction. In brief, it is the power to parallelize light. The power as determined by the posterior focal length, which may be called its posterior power, represents the power of the surface to change, i. e., to converge or diverge incident parallel light.

The formula for the anterior power, the one usually taken, is $D = aM$; for the posterior power, $D = \frac{aM}{n}$. These may be easily remembered as two short words, "dam" and "damn." $D$ in this formula represents power in diopters; $M$ represents curvature in metrecs and $n$ represents the index of refraction of the second medium, the index of the first medium being 1. When the first index is not 1, it must be made 1. For example, if light goes from a substance whose index is 1.33 to one of index 1.42, as from the aqueous into the crystalline lens, one makes the first index 1 by dividing it by 1.33. But then one must also divide the second index by the same number, that is, divide 1.42 by 1.33. This gives as the index of the first medium 1.33 over 1.33, or 1, and that of the second medium 1.42 over 1.33, or 1.07. In other words, $n$ in the formula always represents the so-called relative index, which is the second index divided by the first index. This can be taken as a general rule in all cases. To get $n$, the index to be used in the formula, divide the index of the second medium (to which the light goes) by the index of the first medium (from which the light comes). In this way $n$ may sometimes be less than 1. For example, when light goes from a substance whose index is 1.42 to one of index 1.33, as from the lens into the vitreous humor, the index used is 1.33 divided by 1.42, which gives 0.94. The letter $a$ represents the excess of the second (relative) index over 1; it equals $n$ minus 1. The $a$ is a reminder that it is the amount above 1, the add, so to say. Thus if $n$ is 1.37, $a$ equals 0.37; if $n$ equals 1.04, $a$ equals 0.04, etc. Occasionally $a$ may be negative; thus, if the second (relative) index $n$ is 0.94, $a$ equals 0.94 less 1, which is minus 0.06.

A few illustrations will now serve to show the method of procedure. Suppose one wishes to find the refracting power, anterior power, of the

surfaces of the eye. For convenience, whole numbers will be taken for the radii, such as 8 and 6 mm. for the surfaces of the cornea, and 10 and 6 mm. for the surfaces of the crystalline lens. For the index of the cornea one can take 1.37, for that of the aqueous 1.33, for that of the lens (total or equivalent index) 1.42, and for that of the vitreous 1.33. In the formula $D = aM$, the following figures are substituted; $a$ equals 0.37 (the first index is 1, that of air, and the second index is 1.37, that of the cornea; therefore, 1.37 minus 1), $M$ equals 125 (1,000 divided by 8), and $D$ equals $0.37 \times 125$, equals 46.25 diopters.

For the posterior surface of the cornea, better considered as the anterior surface of the aqueous, one gets similar data. As the light goes from cornea to aqueous, $n$ in the formula is the relative index of the aqueous, that is, the given index of the aqueous divided by the given index of the cornea (second $n$ over first $n$). Dividing 1.33 by 1.37 gives $n$ equals 0.97; $a$ equals this index less 1, which is minus 0.03; $M$ equals 166.7 (1,000 divided by 6). Substituting these in $D = aM$, one gets $D$ equals minus $0.03 \times 166.7$, equals minus 5.00 diopters.

It will be noted that the posterior surface of the cornea has the effect of a concave lens. For the anterior surface of the lens, $n$ equals 1.42 divided by 1.33, which is 1.07; $a$ equals 0.07 (1.07 less 1) and $M$ equals 100 (1,000 divided by 10). $D$ therefore equals $0.07 \times 100$, equals 7 diopters. Finally for the posterior surface of the lens, or rather the anterior surface of the vitreous, $n$ equals 1.33 divided by 1.42 (second index over first index), or 0.94; $a$ equals minus 0.06 (0.94 less 1), and $M$ equals minus 166.7 (1,000 divided by 6). The curvature here is minus, as the surface is concave to the incident light. The result will be plus, as the minus of $a$ is multiplied by the minus of $M$. $D$ equals $0.06 \times 166.7$, equals 10 diopters.

In a similar manner the posterior power of any of the surfaces, and of course the surface and combined power of thin lenses, mirrors, etc., can be calculated. All this is readily accomplished by noting the two simple formulas, which are easily remembered as dam and damn, and by understanding the significance of the four symbols used. In conclusion, I may add that I have seen doctors who had a dread of mathematics and the sight of optical formulas take up this method and master it within an hour or two.

# MECHANISM OF ACCOMMODATION STUDIED EXPERIMENTALLY

MAX F. MEYER, PH.D.
ST. LOUIS

## A HYDRAULIC-OPTICAL EXPERIMENT WITH A FLEXIBLE LENS

Nobody doubts that the lens of the human eye changes its shape in the process of accommodation. Nevertheless the lens lacks any known physiologic properties through the mediation of which it could, in response to stimulation, change its shape spontaneously. A flexible lens, such as that of the human eye, as a mere object of study for the mechanical engineer can change its shape, without ceasing to be a lens, under the influence of any one of three classes of external mechanical factors: (1) by radial pull applied to its circumference, (2) by a contracting push applied to its circumference and acting toward its axis, (3) or by hydraulic pressure applied to one of its two surfaces, provided that the yielding of its peripheral parts is more obstructed than the yielding of its more central parts. (Hydraulics, of course, includes hydrostatics.)

The first two, especially the very first, of these three possibilities have been discussed in the literature of the medical sciences for nearly a century, with numerous arguments for and against. It is utterly strange that the third possibility has been virtually neglected. Mechanical models have been constructed for demonstrating the first two possibilities to students, although none of these has ever been a truly optical model, that is, a model with a lens soft enough to be deformable and transparent enough to make optical observations possible. And the anatomic interpretation of the principles of such mechanical lens models constructed by physicists has generally been unconvincing to those critically inclined, however appealing to laymen.

Three years before the present writing, I began attempts at demonstrating the third possibility of a deformation of the lens, that is, by hydraulic forces. The following pages report the present status of my endeavors.[1] Figure 1 shows the flexible lens in its frame and ready for the experiment. Figure 2 shows the same lens, black in this figure, in the casting process. The method of casting will be described later in this article. The dots appearing within the "ligament" that fastens the lens

1. A preliminary report is: Meyer, Max F.: The True Mechanism of the Accommodation of the Lens, Psychol. Bull. **28**:232, 1931.

to its frame in figure 1 indicate in the model a reenforcement consisting of wire netting. Let it be said at once that this reenforcement is not intended to be the counterpart of anything in particular in anatomy except structural strength of a certain degree. The cavities of the eye model, on either side of the lens, can be filled with a liquid or may be left filled with air.

The cavity on either side can be, but only on the side containing the convex surface of the lens actually is, subjected to varying pressure by means of the bellows shown in the upper part of figure 1. The "eye" here is cylindric, and is composed of metal rings 5 inches (12.7 cm.) in diameter and well fitted together—with the exception of one ring which

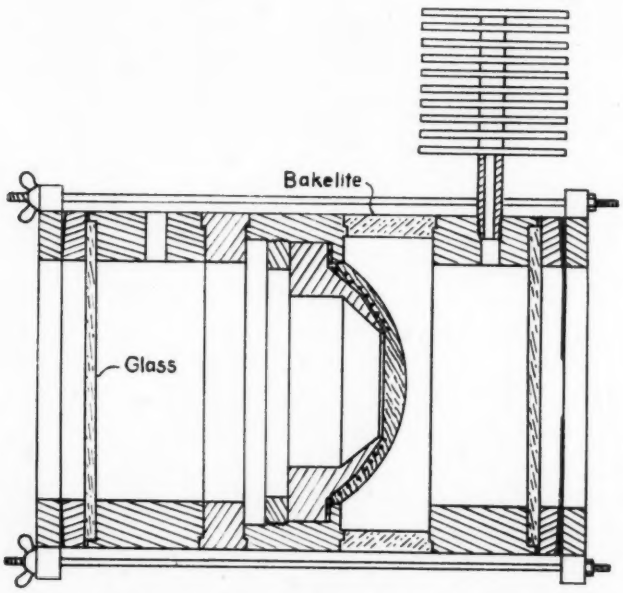



Fig. 1.—The flexible lens in place between two chambers. One chamber communicates with a metal bellows such as is used for steam valve operating; the other chamber communicates with the outside air.

is made of transparent bakelite in order to permit the transverse observation of the deformation of the lens caused by hydraulic pressure. The ends of the cylinder are made of plane glass. The optical diaphragm (or "stop") can be placed on either end, depending on the location of the source of light. Its perforation should not be too small, since a very small perforation would greatly reduce the optical change to be observed; a diameter of about one-half inch (1.2 cm.) is feasible, and of 1 or 2 inches (2.5 or 5 cm.) perhaps even better.

It is advisable to use as a luminous object a clear lamp bulb having a circular filament, and to place the bulb so that the image on the other side of the eye model appears as a bright circle of about the same

diameter as that of the real filament. It is advisable to surround the bulb with a metal box having only one opening, toward the eye model and but slightly larger than the circle of the filament of the lamp, in order to keep the room in which the experiment is performed as dark as possible. Any sheet of white paper may serve as a screen on which to throw the image.

It was the original intention to use a flexible lens with a diameter of 3 inches (7.5 cm.), but since the transparency of a gelatin lens is only moderate, the experiment was finally performed most successfully with a lens with a diameter of only 2 inches. Such a lens can of course be

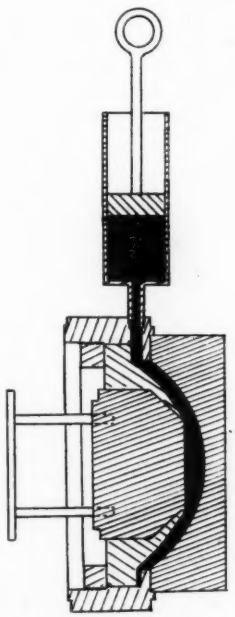


Fig. 2.—The black part of this figure shows the flow of the warm jelly in the process of casting the lens.

made much thinner than a 3 inch lens. For ease in constructing the mold, one of the surfaces of the mold was made plane and the other surface hollow. Accordingly, the lens used in this experiment is a planoconvex lens when at rest. Its focal length in air happens to be about 5½ inches (14 cm.). That is, an object placed at a distance of about 11 inches (28 cm.) from the lens will throw on a screen placed about another 11 inches further a "life-size" image.

It was also the original intention to keep the lens within the eye model in a preserving fluid that would have a density as nearly as possible like that of the lens and a refractive index as different as possible from that of the lens, and to perform the experimental demonstration, whenever desired, in this optical medium. The lens would then be

safe from distortion by gravity in that fluid, even if kept for many years, because it would never sag either downward or upward. But such a preserving fluid is hard to find. When gasoline was used as the liquid to keep the lens in, the focal length was so greatly increased that the experiment could not be performed in a laboratory room of ordinary dimensions without the use of mirrors, and the mirrors available diminished the definition of the image to be observed, which was not very great because of the not yet avoidable imperfections of the casting. Still worse, however, was the circumstance that, when the index of refraction of the lens differed but slightly from that of the medium, observing the direction of the change of the dioptric power was unpleasantly difficult. The gasoline was therefore drained off and replaced by air.

*Experiment.*—The luminous circle is placed at a distance of about 11 inches from the optical center of the lens. The paper screen is shifted until a maximum of definition of the image is obtained. Within a region of nearly half an inch the definition seems to be about equally good because of the imperfections of the lens, which make a very high degree of definition impossible. However, when now the bellows is compressed, the ring-shaped image is so blurred that it looks like a disk rather than like a ring. It is necessary to rearrange the screen, shifting it more than 2 inches nearer to the lens, before a definition of the ring is reached that can be said to be equal to the formerly existing definition.

The experiment, therefore, is convincing. The result can be reliably stated as follows: *An unsymmetric convex lens of flexible material, when held in a frame and brought by one-sided hydraulic pressure nearer to symmetry, draws its effective focal plane closer.*

## ANATOMIC INTERPRETATIONS

Figure 3 is schematic in order to aid the theoretical discussion by leaving out the momentarily irrelevant. In this figure the lens and the vitreous body have both been hatched, though differently, in order to symbolize that in certain mechanical respects these two must be considered as a unit.

That they form a mechanical unit is often forgotten for two reasons: 1. Because they are two in the admitted sense that they are two subdivisions of the unit, of which each has its own structural stiffness. The stiffness of the vitreous body is by no means as great as that of the lens. 2. Because they are two in the optical sense that the refractive index of the vitreous body is almost identical with that of the aqueous humor in front of the lens, and that the lens has a refractive index much higher. Optically the lens stands alone. Mechanically the aqueous humor stands alone.

The ciliary muscle, inserted near the place where the cornea and the sclera join, as shown in figure 3, consists for the most part of

meridional fibers which, contracting, exert a pulling force on the choroid sack (black in the figure), to which the muscle is attached. This sack with all its contents is then inevitably drawn forward, provided two conditions are fulfilled: Something must yield under the posterior sclerotic shell of the eyeball, and something must yield in front under the corneous shell of the eyeball; otherwise the ciliary muscle would merely assume internal stresses without causing any motion. When one looks for something that may yield, one wisely looks for liquids within the eyeball communicating with liquids outside the eyeball. Such liquids are the contents of the blood vessels. The posterior part of the choroid sack is richly supplied with blood vessels. When the

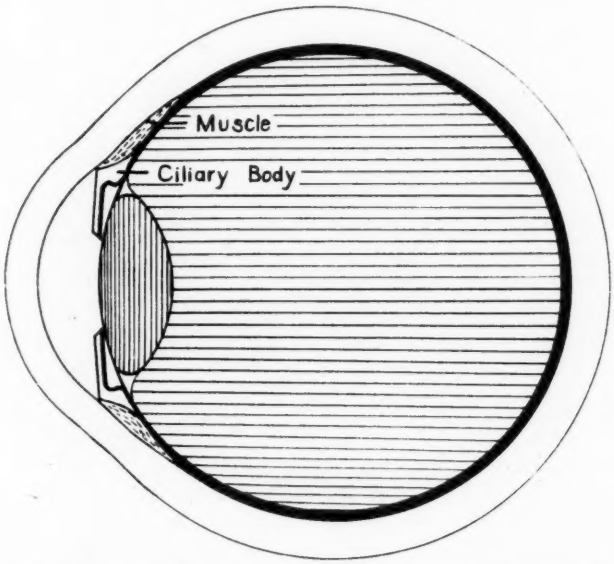



Fig. 3.—The density of the hatching symbolizes the function. In one respect, lens and vitreous body combined are a mechanical unit, a solid in a tapering tube. In another respect, the lens is a flexible shield separating two fluids.

sack with all that fills it is drawn forward, no vacuum need be expected behind. The blood vessels simply gorge themselves with arterial and venous blood, exactly as the lungs fill with air rather than permit a vacuum between themselves and the expanding wall of the chest. So the blood comes on from without and fills the vessels within the choroid sack to completeness.

Something, further, must yield in the frontal chamber of the eyeball when the choroid sack, with the lens-vitreous body caught in it, is pulled forward. The aqueous humor could yield readily if it drained directly into open vessels; but this it does not do. Nevertheless it can yield without any particular difficulty by escaping backward peripherally through the pectinate regions and compressing the numerous ciliary

processes. These are easily compressed, since they are rich in blood vessels and the blood can readily be squeezed out of them backward, that is, out of the eyeball. Essentially, then, it can be said that the blood vessels of the eyeball are the reservoir permitting whatever surging in and out of blood is necessary for the motion of the choroid sack with the lens-vitreous body.

What, then, brings the choroid sack with the lens-vitreous body back to its resting place after innervation of the ciliary muscle has ceased? There is scarcely any special assumption necessary beyond the observation that it behaves like any other body in nature. In science one calls this natural behavior of bodies their elasticity, and means thereby that when they are momentarily strained by external forces they resume their resting shape as soon as those external forces (the ciliary muscle pull in this case) desist from straining them.

To the curiously shaped ciliary processes, like all other details shown only schematically in figure 3, is here ascribed a double rôle. A double or even triple rôle of an organ is nothing strange in biology. Such structural-functional parsimony is not surprising. It is generally believed that the ciliary processes are secretory organs, secreting the aqueous humor. To this function is added another: They are regarded as blood-filled cushions of the aqueous humor, from which this liquid may temporarily squeeze out some blood in order to make needed room for itself, when partially pushed out of its regular place or places.

There arises now a problem (but not an unusual one) of mechanics, which one must solve as an engineer, not as a prejudiced biologist. What happens when a mass which, jelly-like, combines some of the properties of a solid with some of the properties of a liquid is pushed along a tube that tapers forward? I shall first consider what happens to it as a liquid bearing a flexible shield in front. I shall secondly consider what happens to it as a solid, which, like any solid, can be strained out of its shape by being squeezed.

1. While it is true that the lens and the vitreous body are a mechanical unit, shown as such in figure 3, they are nevertheless two bodies when the greater coherence of the front part and the greater liquidity of the rear part are considered. We should say that the lens and the vitreous body together are "a jelly," but that the ratio of gel to sol (speaking in terms of colloidal chemistry) is much higher in the lens than it is in the vitreous body. Relatively, then, the vitreous body behaves like a liquid driven forward by the choroid sack against the lens; and the lens then behaves like a flexible solid, a shield under hydraulic pressure from behind.

The shield suffers in its center only the obstruction resulting from the presence of the aqueous humor, which, as has been shown, can

yield. More peripherally it is obstructed, in addition, by the whole iris including the basal structures of the iris, which are penetrable by a humor from the front, but nowise by any solid from the rear (compare figure 3). It is known that this diaphragm, the iris with its adjuncts, contracts during accommodation, thereby indicating increased muscular resistance. It is known that animals like the otter, which needs a particularly powerful accommodation in order to see under water, have particularly strong iris muscles. The innervated iris muscles tend to present to the lens-vitreous body a funnel into which this body is pressed, and thus to obstruct the periphery more than the center. It is not merely the inner margin of the iris to which I refer. Essentially, what happens is the same as what happens in the model, where the lens is fastened to the frame, but to nothing centrally. Under the hydraulic pressure from behind, the center of the lens, as the least obstructed part, moves forward most, its concentric parts less and less the more peripherally they are located.

The lens in the model is changed from a planoconvex lens into a double convex lens. The lens in real anatomy is double-convex from the start, but like the other it is changed from greater asymmetry to lesser asymmetry, and the law found in my experiment applies.

2. Second, the lens-vitreous body is considered as an undivided mechanical unit which throughout behaves like a solid body, although the rear part of this solid body is not so stiff as its front part, differently hatched in figure 3. As this solid unit, contained in the choroid sack, is pushed into the tapering tube confining it, the effect is similar to that of any kind of wire drawn by the usual method. The only difference is that the wire is pulled, not pushed. The wire, when drawn through a tapering opening in a hard plate, is squeezed together. In the case of metal being squeezed, the elastic limit is soon reached and the material gets a permanent set, which the user of the wire desires. The wire, coming out thinned, remains thin. In the case of jelly, the elastic limits are great. The thinned lens-vitreous body, when permitted to slip back, regains its former size and shape.

It may be worth mentioning that one must not misinterpret the fact that on cutting an eyeball and letting the vitreous body escape, the latter is apt to give one the impression of a viscous liquid running out, like a viscous oil from a bottle in a filling station, and like oil spreading where it falls, rather than that of a bit of jelly dropping on something. One must remember that, as has already been stated, the ratio of gel to sol in this body is very small, so that the gel is easily broken up through deformation. The gel molecules (this is the usual assumption of colloidal chemistry), unless the temperature is too high, tend to form a three-dimensional network within which the sol molecules are confined in droplets. If the body is strained beyond its elastic limits, the small

fraction of gel may easily be broken up so largely that its remaining threads of network no longer seem to confine any of the sol; that is, so that the whole mass runs off like a viscous liquid without even suggesting that a moment before, while still a good jelly and contained within its natural membrane in the eyeball, it nevertheless possessed the mechanical properties of a solid body to a considerable degree.

When the wire, being drawn, is thinned, it lengthens. When the lens-vitreous body is thinned axipetally, it lengthens axially. All of it lengthens axially, although it is true that the rear part, being less stiff, thins and lengthens more than the front part, which is stiffer. Optically, however, the thinning and lengthening of the rear part might almost be forgotten because its index of refraction is virtually identical with that of the liquid filling the frontal chamber of the eye and thus is merely "an optical medium" instead of being considered as an optically active agent. On the other hand, the axipetal thinning and axial lengthening of the front part are not to be forgotten optically, since this part has the property of a highly refractive lens. (The rear part, however, may thin more, with the result that in extremely powerful accommodation the membrane called the zonula becomes curly and the lens may easily tremble, as has been frequently observed.)

A convex lens that contracts axipetally and lengthens axially reduces its focal length. Therefore one has here a second effect of the nature of accommodation for nearness, like the first effect brought into being by the pull of the ciliary muscle on the choroid sack.

I shall now consider changes 1 and 2 together, both architecturally and optically. According to the first, the lens is a flexible shield the center of which is hydraulically pushed forward, so that both poles of the shield would move forward. According to the second the frontal pole of the lens would move forward more than its rear pole would in the thinning and lengthening process.

The total forward movement of the frontal pole of the lens thus must be more conspicuous than the total forward movement of the rear pole. The latter is difficult to measure in the living eye, unless one invents a method of inferring it logically. But the logic of these inferences is not reliable. Nevertheless, there are no facts known that contradict the statement made. Further, no data are known from which any estimate can be derived of the separate amounts of the architectural changes 1 and 2. Here one is still in a field of speculation.

Optically it is clear that, since change 1 by experiment means near accommodation and change 2 by consensus means near accommodation, both together mean even more efficient near accommodation. Whether the first effect contributes more than the second effect, or the second contributes more than the first, nobody knows. In other words, nobody

knows whether the hydraulic effect contributes more to near accommodation than the axipetal ("wire-drawing") effect or the reverse.[2]

### THE CASTING OF A FLEXIBLE LENS

When a few years ago it became clear to me that the problem of the mechanism of accommodation could not be solved without experiments on artificially constructed flexible lenses, I cast about for information on how to obtain flexible lenses, without suspecting the disappointment that was in store for me. I could not discover any one who had experience in making flexible and transparent lenses, although I received from workers with gelatin and similar substances some hints as to how this might be done. I started experimenting, and gradually improved the method from an almost hopeless one to a process that holds out promises of further improvement. I shall describe how my present lens was cast. It was the fourteenth attempt on my part.

A flask is placed on a balance and found to weigh 6½ ounces (184.2 Gm.). High grade "de-ashed" gelatin, obtained from the Eastman Company of Rochester, N. Y., is cut with scissors into strips narrow enough to pass through the neck of the flask. The flask is then filled with them until it registers 8 ounces (226.8 Gm.) or 1½ ounces (42.5 Gm.) of gelatin. Enough distilled water, of room temperature (not warm), is then poured in to cover the gelatin strips; and the flask is left alone for four or five minutes to permit the gelatin to swell. The water is then poured off and the flask weighed again. It now weighs 10¼ ounces (290.6 Gm.), so that the gelatin is known to have absorbed 2¼ ounces (63.8 Gm.) of water. (If more is desired, one can still add more water, but if the dry gelatin is allowed to swell too much, the mistake is not so easily remedied.) An ounce

2. A few historical notes may here be added: ". . . one preposterous assumption seems to stand in the way . . . that is the assignment of an impossible rôle to the physical property of matter called 'elasticity.' It is foreign to the true meaning of the word . . . to assume that elastic tissue is maintained in a state of deformity while at rest" (Luedde, W. H.: The Mechanism of Accommodation: Facts and Fancies, Arch. Ophth. **7**:66 [Jan.] 1932). In the following discussion the popularity of this preposterous assumption was traced to its root by P. C. Kronfeld (Arch. Ophth. **7**:69, 1932): ". . . the theory . . . may be explained to lay people in a minute." To this true and trite statement I would add the following remark: "And students can learn it by rote. But if a physiologic theory can be explained to laymen in a minute, we men of science ought to be extremely critical of it."

The foregoing preposterous assumption misled Helmholtz not only in ophthalmology, but also in otology, in which he assumed "crosswise tension, at rest, in the basilar membrane." (Compare "anchoring versus stretching" [Meyer, Max F.: The Hydraulic Function of the Cochlea, J. Gen. Psychol. **1**:240, 1928]). Quandoque bonus dormitat Homerus. By the way, the "ingenious Helmholtzian theory," as one author calls it, or "preposterous assumption," as Luedde called it, of lenticular elasticity did not originate with Helmholtz, but with William Bowman, in 1847, when he published an article in the Philosophical Transactions of London on the ciliary region of the eyeball.

(28.3 Gm.) of pure glycerin is now added, and the contents of the flask are melted in a water bath below the boiling point of water. It must be remembered that too high a temperature or too prolonged heating permanently reduces the ratio of the gel to the sol, and that seems undesirable.

When the whole is well dissolved and mixed, it may be filtered hot through white woolen flannel. Filtering improves the transparency of the final product. But if one has no suction filter handy, the process is not feasible. Even without filtering, one can obtain a fair degree of transparency.

In a little separate vessel a preparation is kept ready which consists of half an ounce (14.1 Gm.) of glycerin in which 16 drops or about 0.5 cc. of 40 per cent formaldehyde has been dissolved. This will be referred to as glycerin-formaldehyde.

The mold is prepared as shown in figure 2. The central metal ring of the eye model is separated from the other rings, and the pumping cylinder with its piston is added. The bronze molds, one of them plane and the other a hollow segment of a sphere, are carefully polished on the surfaces against which the casting will later lie. These surfaces are then treated with a rosinous furniture polish which covers the metal, since metal would be attacked by the formaldehyde and the jelly would stick to the molds when the latter were removed.

The lens molds are then put against the metal ring and clamped in place. The whole is then kept for a while in a low temperature oven in order that the jelly, when being poured, may remain hot and easily flowing and may distribute itself easily within the narrow-spaced mold. A wide open glass vessel having a spout from which one may pour the contents in a narrow stream is then heated (below the boiling point of water), the gelatin solution is poured from the flask into this vessel, and the glycerin-formaldehyde is added and carefully, although without delay, stirred in. It is difficult to avoid, and yet most important to avoid, the formation of bubbles. If the formaldehyde is added or distributed too slowly, it may cause the jelly to set in lumps or to set as a whole before one has had time to get it into the mold. Only two or three minutes is available.

The hot mold is now taken from the oven. The gelatin solution as treated with formaldehyde is carefully poured into the cylinder above the mold, as shown in figure 2. The piston is inserted and pushed down with just enough force to squeeze out the gelatin gently in from ten to fifteen seconds. Two or three needle holes in the periphery of the metal ring, judiciously placed, permit the escape of the air from the mold. They are closed with phonograph needles as soon as the gelatin is beginning to well out from them. The whole mold, including the piston handle, is now placed in a refrigerator or simply left alone for the gelatin to gel. In figure 2 the gelatin appears black.

The cylinder, with the piston and some hardened gelatin within it, is easily removed by unscrewing it. The jelly thread formed in the connecting piece simply breaks off. The hollow piece of the lens mold is removed by means of a screw which tends to lift one side of its rim. It is important to apply little force with this screw, and this only at intervals of several minutes each, in order that the cast jelly will have time to loosen itself from the surface of the hollow mold without tearing. The plane mold is separated more easily from the cast jelly, but nevertheless must also be removed with care.

The metal ring holding within it the finished jelly lens is now joined with the other rings and parts of the eye model as shown in figure 1. The metal bellows may be replaced by a funnel, if it is desired to fill the chamber with a liquid. The same funnel may be applied to the other chamber to fill this also with a liquid.

The idea readily occurs to add a highly refractive salt to the gelatin solution before casting it. This would make the optical demonstration spectacular. Unfortunately, the presence of any salts more or less prevents the gelling of the solution in convenient temperatures; it reduces the ratio of the gel to the sol. It goes without saying that it is inconvenient to perform optical experiments within a refrigerator.

It might be possible to gel the solution sufficiently for mechanical experiments without stirring in any watery formaldehyde solution, by solidifying the jelly lens first without formaldehyde and then giving the lens already formed the protective treatment through exposing it long enough to mere formaldehyde gas. The casting of a perfect lens would be greatly aided if that were possible. I have not had the facilities for trying that method.

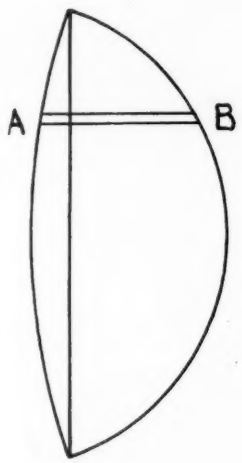



Figure 4

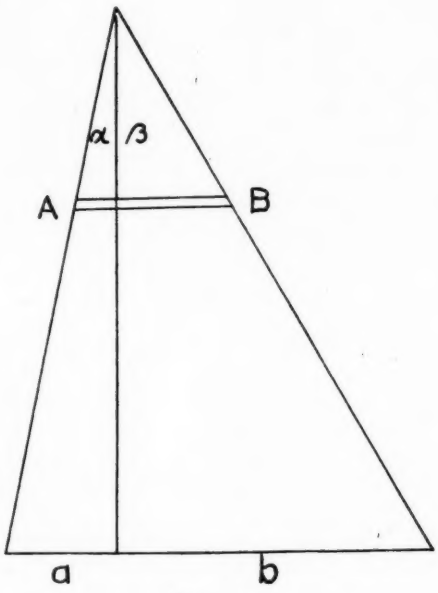



Figure 5

Fig. 4.—The lens thought of mechanically as if it consisted of infinitely many, infinitely thin and parallel sticks or prisms. This mechanical idea may be wrong.

Fig. 5.—Tangents of the lens surfaces and their angles. The decrements or increments of these angles are by no means equal, except in the neighborhood of symmetry.

Gasoline would be an ideal liquid for filling the chambers if its refractive index differed more from the refractive index of the jelly lens. When gasoline is used, the joints of the eye model can easily be made liquid-proof by the same mixture used for casting the lens, only leaving out the formaldehyde. To take the chambers of the model to pieces for any purpose, one need only put the glass and metal parts in fairly hot water for a few minutes, which makes the "cement" quite soft. (Warning: The transparent bakelite may begin to soften undesirably when placed in boiling hot water.)

I made one unsuccessful attempt at filling the chambers with a liquid of a refractive index much higher than that of the jelly lens. I used carbon disulphide.

This, however, deposited carbon particles all through the mass of the lens, and after a few weeks the lens was as black and as opaque as ebony.

Chemists of the Du Pont laboratories advised me that it might be possible to cast a lens of good transparency combined with sufficient flexibility, using one of the various kinds of cellulose. I have not had the facilities to try this out.

### A POSSIBLE THEORY OF THE DEFORMATION OF THE LENS

To give at this moment a complete mathematic-optical theory of deformation of the lens is not to be expected of any one. For that, experimental data are needed which I do not possess because hitherto nobody has experimented with solid and flexible lenses, either mechanically or optically.

Let the reader consider merely what would happen to the curvatures of the two lens surfaces under the assumption that all the molecular displacements would occur strictly parallel to the axis of the lens. This assumption can also be worded by saying that the lens, for mathematical purposes, might be thought of as a collection of infinitely thin prisms, all lying parallel to the axis of the lens, each of these prisms being capable only of sliding along its own axis and not capable of any other motion or of any shortening or lengthening. This is suggested by figure 4, in which $AB$ is such a prism, except that it cannot be drawn infinitely thin.

Figure 5 shows the same prism between $A$ and $B$, but it shows also auxiliary lines, two of them being tangents geometrically to the lens surfaces and one a parallel to the axis of the lens, making it possible to draw conclusions through the application of mathematical theorems.

It is obvious that in figure 5, if for ease of statement the line drawn normal to the axis is given the numerical value of a unit, line $a$ is trigonometrically equal to the tangent of $\alpha$ and line $b$ is trigonometrically equal to the tangent of $\beta$.

The simplest mathematical procedure now is by the application of the calculus. The differential quotient of $a$ is found for the variable $\alpha$, and the differential quotient of $b$ for the variable $\beta$.

$$\text{I} \quad \frac{da}{d\alpha} = \frac{d \tan \alpha}{d\alpha} = \frac{1}{\cos^2 \alpha}$$

$$\text{II} \quad \frac{db}{d\beta} = \frac{d \tan \beta}{d\beta} = \frac{1}{\cos^2 \beta}$$

In the equations I and II the expressions $da$ and $db$ are the increment and the decrement of one prism when pushed forward under hydraulic pressure and under the assumption already made. The values of $da$ and $db$ are necessarily equal, for the prism in motion cannot lose more behind than it gains in front. Under these circumstances $da$ and

*db*, being equal, can be made to disappear from the equations by dividing II by I; the following new equation results:

$$\text{III} \quad \frac{d\alpha}{d\beta} = \frac{\cos^2 \alpha}{\cos^2 \beta}$$

Equation III is easily interpreted. It states that the decrement or increment of either angle is by no means equal to the decrement or increment of the other angle, except in the neighborhood of symmetry of the lens, that is, when $\alpha$ and $\beta$ are virtually equal. But off symmetry, when $\alpha$ and $\beta$ are different, the larger angle (whichever it may be) loses (or gains) enormously less than the smaller angle gains (or loses), namely, in proportion to the *square* of its cosine. The less symmetrical the lens, the greater this disproportion of loss and gain under hydraulic pressure, because two cosines differ considerably only when their arcs differ considerably, and the squares (second power) then exaggerate this difference. *That is exactly why the lens in the eye ought to be, and is by evolution, asymmetric in its very state of rest.* Whenever hydraulic pressure now interferes with its state of rest, whenever it pushes the rear surface and reduces its curvature, the front surface gains vastly more in curvature than the rear surface loses.

That this disproportionate change in curvature is an explanatory feature of prime importance will appear plausible to the reader. Nevertheless, it is not asserted that this is an exhaustive mathematical treatment of the optical problem of flexible lenses. Still it makes one feel less bewildered at the outcome of the experiment, in which I discovered the fact, expressely denied by Helmholtz, although he guessed at it, that the mere concentric flexing of a lens reduces its effective focal length.

# GROUPED PIGMENTATION OF THE RETINA

WILLIAM A. MANN, JR., M.D.
CHICAGO

This definite but rarely described clinical entity, called by various observers "melanosis of the retina," "congenital pigmentary plaques of the retina," "congenital pigmentation of the retina," and, in an excellent review of the literature by Hoeg [1] in 1911, "grouped pigmentation of the retina," is probably more common than the relatively few recorded cases would lead one to suspect. Thus in seven years Hoeg saw four cases; Blake [2] found seven cases in twelve years, and Stephenson [3] observed the condition three times in 2,400 examinations. The three cases here described were seen in a single year; S. R. Gifford has observed two other previously unreported cases. In spite of this percentage there is very little in the literature regarding this interesting congenital anomaly, and relatively few cases have been described. Most textbooks on ophthalmology make no mention of it, although Ball [4] listed it, unnamed, under "variations in retinal pigment" in the normal fundus, and illustrated it by a typical drawing of the condition. A brief clinical and microscopic description is also given by Collins and Mayou [5] under the title "congenital pigmentation of the retina."

The first case of grouped pigmentation of the retina was described in the early days of ophthalmoscopy by Mauthner [6] (1868), and was published by Jaeger [7] in his "Ophthalmoskopischer Hand-Atlas" in 1869. These authors described it as a "typical pigmentary change," although they did not state whether other similar cases had been observed, and it is to be inferred that they regarded it as an atypical

From the Department of Ophthalmology, Northwestern University Medical School.

1. Hoeg, Niels: Die gruppierte Pigmentierung des Augenhintergrundes, Klin. Monatsbl. f. Augenh. **49**:49, 1911.

2. Blake, E. M.: Congenital Grouped Pigmentation of the Retina, Tr. Am. Ophth. Soc., 1926, p. 223.

3. Stephenson, Sidney: Tr. Ophth. Soc. U. Kingdom **11**:77, 1891.

4. Ball, J. M.: Modern Ophthalmology, Philadelphia, F. A. Davis Company, 1926, vol. 2, p. 776, plate xx, fig. 1, opposite p. 822.

5. Collins and Mayou: Pathology and Bacteriology of the Eye, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1925, p. 27.

6. Mauthner: Lehrbuch der Ophthalmoscopie, Vienna, Tendler & Company, 1868, p. 388.

7. Jaeger, Edward: Ophthalmoskopischer Hand-Atlas, Vienna, K. K. Hof und Staatsdruckerer, 1869, fig. 76.

form of retinitis pigmentosa, with which it is now known to have no relationship. Subsequent cases were added by Jacobi [8] (1868), Frost [9] and Stephenson (1889-1891), Segalowitz [10] (1906), Hoeg (1911), Tamamscheff [11] (1911), Kraupa [12] (1917 and 1921), Hedde [13] (1920), Holm [14] (1921), Blake (1926) and others. Kraupa stated in 1921 that there were only fifteen cases in the literature, and a few have been added since that time. In 1926, Blake found references to twenty-eight cases and added six more.

The anomaly is characteristic in appearance and once seen will never be forgotten. It is characterized by a grouping together of spots of pigment in the retina, from three to thirty in a clump, of varying sizes but all sharply outlined, more or less regular, roundish or oval, mostly uniformly grayish black, rather punched out in appearance and not fading into the remaining (normal) fundus. In size they vary from less than the diameter of a blood vessel up to the diameter of the papilla, the smaller spots usually lying near the disk, the larger farther out in the periphery. Only one portion or segment of the fundus may be involved; some cases are unilateral, others bilateral. The macula is always spared. Most observers agree that the pigment lies behind the level of the retinal blood vessels, although Holm was certain that many of the spots were in front of the vessels. The condition appears to be nonprogressive and is assumed to be congenital, although the youngest patient observed was an infant 8 months of age.[2] There is no effect on the central visual acuity or the visual fields. Other congenital anomalies, such as epicanthus, congenital cataract in the other eye and strabismus, have been observed in some of the recorded cases; most of them, however, are otherwise quite normal. No night-blindness has been observed. Consanguinity, hereditary syphilis and sex seem to play no etiologic rôle. There are no evidences of inflammatory or degenerative change, and the course and appearance differ so radically from retinitis pigmentosa and chorioretinitis as to make confusion impossible.

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8. Jacobi, S.: Pigmentmassen in der Retina ohne Störung der Sehfunktion, Arch. f. Ophth. **14**:1 and 144, 1868.

9. Frost, W. A.: Brit. M. J. **2**:1319 (Dec. 14) 1889.

10. Segalowitz: Ophth. Klin. **10**:225, 1906.

11. Tamamscheff: Gruppierte Pigmentierung des Augengrundes, Vestnik Opth. **10**:776, 1911.

12. Kraupa, E.: Studien über die Melanosis des Augapfels, Arch. f. Augenh. **82**:67, 1917; Beitrag zur Morphologie des Augenhintergrundes, Klin. Monatsbl. f. Augenh. **67**:15, 1921.

13. Hedde: Gruppenformige naevoide Pigmentierung der Netzhaut, Klin. Monatsbl. f. Augenh. **64**:301, 1920.

14. Holm, Eijar: Zwei Fälle von gruppierte Pigmentierung, Klin. Monatsbl. f. Augenh. **67**:451, 1921.

There has been considerable confusion in the literature regarding the proper nomenclature for this anomaly. Segalowitz' view, that the pigment is in the choroid, has not been taken seriously by other authors. Kraupa selected the name "melanosis retinae" because it gave indication of no anatomic diagnosis and decried Hedde's use of "naevoid pigmentation," quoting Fuchs as saying that one could speak of a nevus only if nevoid cells were proved. "Grouped pigmentation of the retina," proposed by Niels Hoeg, is still to be accepted, although Blake in 1926 urged that the name be lengthened to "congenital grouped pigmentation of the retina" to emphasize the more or less proved congenital factor.

In theorizing on the pathogenesis of this condition, Hoeg believed that it was an anomaly that appeared in an early stage of development of the eye and was produced by an irregularity in the production of pigment. Sir Herbert Parsons [15] examined one eye histologically and found collections of deeply pigmented epithelial cells in the retina, some of which surrounded nodules of hyaline material, similar to that forming Bruch's membrane. An abnormal union of two layers of the secondary optic vesicle was believed by him to be responsible for the condition found in grouped pigmentation of the retina.

The following cases are entirely typical. All three happened to occur in males, aged 12, 29 and 18 at the time of the first examination. In two the condition was unilateral, in the third bilateral. In none was there any congenital or developmental anomaly, or any effect on the vision, all being discovered during the course of a routine examination of the fundus. Two of the patients were reexamined approximately one year after they were first seen, and no change in the picture could be detected. The cases are illustrated with photographs of the fundi.

## REPORT OF CASES

CASE 1.—F. B., a white man, aged 29, entered the eye dispensary at Northwestern University on Sept. 3, 1930, with a history of frontal headaches and some tiring of the eyes after near work. He had never worn glasses. Vision in the right eye was 20/20 + 4, and in the left, 20/20 + 2. A refraction under homatropine cycloplegia was done. Vision in the right eye with + 0.75 with + 0.50 cyl. 80 = 20/15 —4; that in the left eye, + 0.75 with + 0.50 cyl. 100 = 20/15. At a subsequent postcycloplegic test this correction, less + 0.25 sphere, was ordered with relief of symptoms.

Examination of the fundi at the time of the homatropine refraction revealed no pathologic changes in either eye except for a typical grouped pigmentation of the retina in the nasal half of the left eye. There were numerous groups of varying sized pigmented spots arranged in clusters of from four to thirty closely lying, irregularly sized, roundish or oval dots, situated chiefly along the retinal blood vessels, the smaller ones being about the diameter of the larger vessels, the larger ones, up to three or four times that size. They showed a rather uniform grayish

15. Parsons, J. H.: Tenth Cong. internat. d'opht., 1904, p. 152.

black color, with distinct, punched-out margins. The spots were unevenly distributed between the disk and the nasal periphery, extending above and below. There were no areas of inflammatory or atrophic change. As to the depth of the pigment, all spots seemed to lie under the vessels. Owing to the pigment showing through the vessels, at first it was thought that some spots might lie in front of the vessels, but this was demonstrated by means of the Gullstrand slit-lamp to be untrue.

A general physical examination showed no pathologic changes, and all laboratory tests gave negative results. No family history of ocular trouble or consanguinity was given. The parents wore glasses for correction of presbyopia only. There were three sisters, two of whom wore glasses, but the oculist who fitted them had no record of retinal findings.

Fig. 1 (case 1).—The left fundus oculi, showing typical pigment spots of grouped pigmentation of the retina.

The patient was seen again one year after the first examination, at which time a careful examination of the fundus was again made, and no change was found from the previous situation.

CASE 2.—R. R., a white boy, aged 12 years, was seen through the courtesy of Dr. Richard Gamble; this case is reported with his permission. The patient had been admitted to the Childrens' Memorial Hospital in Chicago, impaired vision in the right eye being his only complaint. Refraction under atropine cycloplegia revealed a hyperopic astigmatism, a + 1.00 cylinder improving his vision in the right eye from 20/40 to 20/20, and a + 0.50 cylinder in the left eye giving him vision of 20/20.

Both fundi were normal except for the nasal half, in which were studded grayish black, pigmented areas arranged in clumps of from three to thirty, with about the same size and arrangement as those in the previous case. The fundi in the nasal portion showed the typical "bear track" appearance of grouped pigmentation of the retina. The pigment spots were quite distinct and homogeneous,

Fig. 2 (case 2).—The right eye, showing involvement of the nasal half. The nasal portion of the left eye was similarly affected.

Fig. 3 (case 3).—The nasal portion of the left fundus oculi, showing grouped pigmentation of the retina.

extending far to the periphery and not involving the macula. All spots lay behind the level of the retinal vessels. The intervening fundus appeared perfectly normal.

A complete physical examination revealed no other pathologic changes. There was no consanguinity, nor a history of ocular trouble in the parents, brothers or sisters.

The patient was reexamined after one year, and no change in the condition was observed.

CASE 3.—J. M., a white boy, aged 18, a university student, entered the clinic at Northwestern University with no ocular complaint except poor distant vision for which he was wearing a low myopic correction. Homatropine refraction was done, his corrected vision being 20/20 in each eye with a slight change in his old lenses. All external and fundus examinations revealed no pathologic changes with the single exception that there was a definite grouped pigmentation in the nasal half of the left fundus. This appeared in the form of from eight to ten patches between a point about 1 papilla diameter nasal to the disk and several papilla diameters nasal to that point. There were from three to thirty dark gray, pigmented dots in each patch, their appearance being identical to those in other cases described.

In this case also no consanguinity or other pathologic condition was found.

## SUMMARY

Grouped pigmentation of the retina is probably much less rare than the attention given it in textbooks and case reports would indicate. The condition should be described under a single name, that suggested by Hoeg being the most acceptable. Three cases are herewith added to the literature, together with mention of Gifford's two cases. The photographs of the fundi presented are believed to be the first published showing this condition.

# FOREIGN SUBSTANCES INJECTED INTO THE VITREOUS OF THE RABBIT

## PATHWAY OF TRANSPORTATION FROM ONE EYE TO ITS FELLOW; AN EXPERIMENTAL STUDY

ERNEST F. KRUG, M.D.
AND
GEORGE L. ROHDENBURG, M.D.
NEW YORK

In spite of the many contributions to our knowledge, the mechanism of the condition known clinically as sympathetic ophthalmia is still a matter of controversy. Consideration of the anatomic and physical factors involved would lead one to conclude that the path of the inflammatory reaction lies from one eye to the other by way of the optic nerve and chiasm. Simple as this explanation may appear, it apparently has been difficult to demonstrate definitely its accuracy.

Before proceeding to the presentation of experimental data gathered during the past two years, we shall briefly survey our present knowledge. This is covered, we believe, by three outstanding presentations since 1924, when Mariani [1] reported the results of the inoculation of three rabbits with the scrapings from herpetic keratitis. In the first rabbit there developed on the sixteenth day in the fellow eye inflammation of the conjunctiva and cornea; in rabbits 2 and 3 similar but less severe inflammations of the other eye developed on the seventeenth and eighteenth days, respectively. Histologically, a binocular perineuritic process was demonstrable in both optic nerves and could be traced along the perioptic lymph tracts and the arachnoidal spaces up to the chiasm.

In 1927, von Szily [2] illustrated a horizontal section through both optic nerves, the chiasm and adjacent brain areas, demonstrating the path of infection from eye to eye (in rabbit 17) sixteen days after inoculating the right eye and three days after the onset of ophthalmia

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From the Achelis Laboratory of the Lenox Hill Hospital.

1. Mariani, G.: Experimentelle Untersuchungen und kritische Erwagungen über die Aetiologie der Herpeserkrankungen, Arch. f. Dermat. u. Syph. **147**:259, 1924.

2. von Szily, A.: Ergebnisse der Infektionsüberleitung von Bulbus zu Bulbus mit Herpes Virus—experimentelle sympathische Ophthalmie beim Kaninchen—unter besonderer Berücksichtigung des Impfmodus, der Uebertrittswege und der Spatstadien, Klin. Monatsbl. f. Augenh. **78**:11, 1927.

in the left eye. He demonstrated that histologically the infiltration extended from eye to eye along the optic tract. The primarily affected side was more markedly infiltrated, but the second optic nerve showed continuous and somewhat less intensive involvement from the chiasm to the second eye. The histologic specimen from another rabbit previously saturated by intravenous injections of trypan blue, with the exclusive and abundant blue cells found in both optic nerves, demonstrated activation of the adventitial cells and inflammation progressing along the optic tract.

Deutschmann,[3] in an article largely a controversial comment on von Szily's paper, quoted his earlier experiment of typical experimental sympathetic ophthalmia of the fellow eye in monkey 2. No clinical general symptoms or any meningitis was demonstrable, and anatomic findings were entirely normal; yet microscopically the entire optic nerve tract showed definite inflammatory phenomena, demonstrating the path of infection from eye to eye. Deutschmann likewise quoted his case, published in 1893,[4] which presented *intra vitam* sympathetic ophthalmia of the left eye. Death occurred from gastric carcinoma. At autopsy, the meninges, the brain and ciliary nerves were found to be normal, while both optic nerves and the optic chiasm showed marked round cell infiltration and clearly demonstrated the optic tract as the path of infection from eye to eye.

In planning the present experimental work, it was thought advisable first to consider the anatomic and physical factors that might be involved in the process. One of the first points to be considered was that of a possible anatomic pathway between the two eyes. Those who question the existence of lymph channels in the optic nerve or chiasm have not considered that the lymph system in its finest subdivisions consists not only of definite channels lined by a layer of endothelium, but also of tissue spaces or clefts between individual cells or bundles of cells or cell fibers. Under high magnification these spaces are frequently seen to contain a hyaline substance (coagulated lymph?) which has been precipitated *in situ* by the fixation of the tissues. Furthermore, wandering cells, such as polymorphonuclear leukocytes, round cells, plasma cells, etc., in small numbers are frequently found normally in these localities and in increased numbers in chronic inflammatory reactions. Such spaces are exceptionally abundant in the sheaths of blood vessels even in those vessels of small diameter.

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3. Deutschmann, R.: Ueber Infektionsüberleitung von Bulbus zu Bulbus, Arch. f. Ophth. **119**:347, 1927.

4. Deutschmann, R.: Fortgesetzte Versuche und Untersuchungen über die Ophthalmia migratoria, Beitr. z. Augenh. **1**:1, 1893; cited by Schirmer, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Englemann, 1905, vol. 6, pt. 2, chap. 8.

Considered in detail, the optic nerve is formed by a massing of nerve fibers about a central blood vessel. Schematically considered, the central blood vessel represents a core from the wall of which radiate spokes of a supporting connective tissue. These spokes act as a support and binding for the nerve fibers. When the nerve is cut longitudinally, this connective tissue framework is demonstrable as long, more or less connected and interlacing fibers. Occasionally definite spaces can be demonstrated in these fibers and still more occasionally these spaces can be shown to be lined by a single layer of flattened cells which have all of the histologic characteristics of endothelium. As the chiasm is approached, it is found that some of these connective tissue

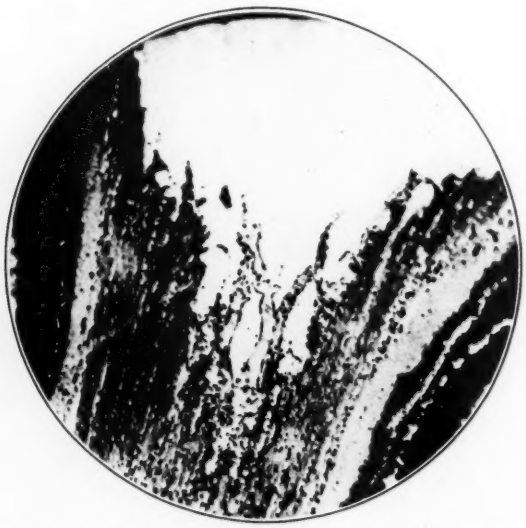

Fig. 1.—Nerve head within an eye showing the blood vessels and the beginning of the lymph clefts.

fibers proceed laterally into the chiasm and are in close relationship, if not in actual communication with similar fibers from the opposite nerve. Others of the fibers continue along the course of the nerve and are ultimately lost in the supporting glia of the brain. These histologic characteristics are shown in the accompanying photomicrographs. Figure 1 (stained by the Maresch Bielschowsky technic) shows the first portion of these fibers at the nerve head within the eyeball. Figure 2, stained with the same technic, shows the fibers in the nerve proper together with the lymph clefts and spaces. On cross-section of either the nerve or chiasm these connective tissue fibers are shorter and their connection with one another is not evident. They appear more as short connective tissue fibers which apparently have no relation with one another.

Having demonstrated that there are pathways that might be followed from one eye to the other via the chiasm, the next point was a consideration of the possible modes of transportation along the demonstrated pathway. There being no reason to suppose that the mechanism of transportation in the eye differed from that observed in any other tissue of the body, it was theoretically proposed that transportation might occur either by the flow of tissue liquids along the clefts, or by the carrying of particulate substances by the wandering cells in a manner similar to the transportation of carbon particles from the alveolar epithelium of the lung to the mediastinal lymph nodes. Our subsequent experiments were based on this hypothesis.

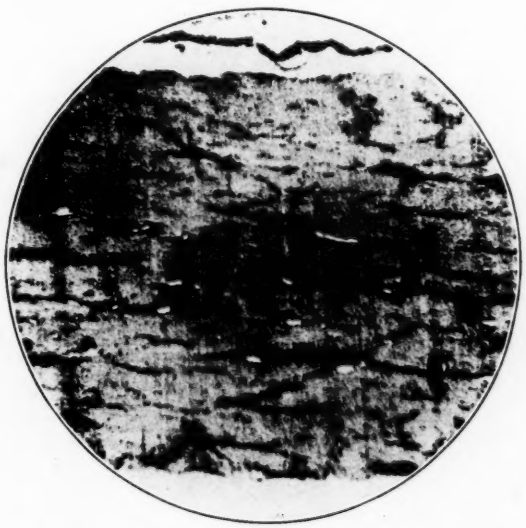

Fig. 2.—Longitudinal section of an optic nerve showing the lymph spans and channels.

In a manner to be described in a later paragraph, foreign bodies of various types and character were introduced into the vitreous of the rabbit eye. After varying intervals the animals were killed and histologic examinations were made of the eyes and the attached nerves—the eyes into which injection had been made as well as the controls. The substances selected were (*a*) india ink; (*b*) a weak suspension of kieselguhr (infusorial earth); (*c*) old tuberculin; (*d*) living bovine tubercle bacilli; (*e*) living Streptococcus hemolyticus, and (*f*) oils of various viscosities, some of which were pigmented with a dye. With the exception of the bacteria, all of the substances were injected in a sterile condition.

The substances selected were chosen for definite reasons. The vitreous is a hydrophile colloid gel, which in the chemical sense may or

may not be reversible, a point that has not yet been determined. If an insoluble foreign body in an exceedingly fine state of subdivision is placed into such a gel, it may be carried out of the gel either by fluid currents or by wandering cells. Whether or not the wandering cell takes up the foreign body is in turn dependent on the chemotactic power of the foreign substance. If the foreign substance has a positive chemotactic influence, it will be taken up. If, however, the foreign body is negative from the chemotactic standpoint, the wandering cells will not engulf and remove it. For the present purposes, india ink, which is a fine suspension of carbon, may be considered as positively chemotatic, while kieselguhr is much less so. Finally, it is the tendency of any tissue to encapsulate more or less rapidly any foreign particle lying within it.

Neither india ink nor kieselguhr has a motive power of its own, and if transported from the site of injection, it must be carried either by wandering cells or by fluid currents. The viscosity of india ink is such that a considerable force must be granted to any current flow that might carry it. Both india ink and kieselguhr are chemically inert and insoluble.

The proteins selected, old tuberculin, living tubercle bacilli, living streptococci, are alike in that all exert a chemical action on the tissues, which chemical action is classified from the histologic standpoint as an inflammation. The living bacteria are particulate, as are the foreign bodies, but they are infinitely smaller than the particles of either india ink or kieselguhr. All three substances might be transported as outlined for the inert foreign bodies, but in addition the living bacteria are capable of a slight motion of their own.

The oils selected were chosen because it was supposed that in the vitreous they would be chemically inert and unaffected by the ocular fluids. They were tinted by a dye, so that the droplets might be more easily found, and they had the ability not possessed by the other substances chosen of being able to change their position under the influence of gravity or of some force applied to them, such as pressure of the surrounding media, etc.[5]

It is not our purpose in the present report to consider or to describe the inflammatory reactions observed, except so far as they might indicate a path from one eye to its fellow eye, or might indicate certain fluid currents.

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5. The injections were made under careful aseptic precautions, an insulin syringe with a dental type hypodermic needle being used; the needle was plunged into the eyeball between the superior rectus and internal rectus muscles, 5 mm. back of the limbus. The eye was desensitized with 2 per cent holocaine solution and the penetrating needle was seen in the vitreous with the ophthalmoscope. One minim of each suspension was injected, and daily examinations of the eyes showed that the drop was gradually breaking up in the vitreous and was migrating toward the retina and back to the nerve head.

OILS

While the changes observed after injection of oil into the vitreous have been described elsewhere,[6] it was deemed advisable again to present briefly the experimental data so far as it concerns the demonstration of a pathway between the eyes. It was originally supposed that oils would be chemically inert in the vitreous, but it was discovered that the vitreous contains a lipase of marked activity. In consequence of the lipase activity, the oil rapidly disappears from the vitreous owing to digestion. If the oil is pigmented by a dye such as scharlach R, this dye is precipitated when the oil has been digested. Since the oil has no chemotactic attraction for the wandering cells, this mode of transportation may be dismissed from consideration until the dye has been precipitated by the digestion of the vehicle in which it is suspended. Transportation therefore would be in greater part due to the force of intra-ocular currents.

Fig. 3.—Oil droplets in the retina. (The poor definition of this and some of the subsequent illustrations is due to the fact that the photomicrographs are of unstained frozen sections.)

On the basis of the observations made in the group of rabbits given injections of various oils, the pathway from one eye to the other as followed by the intra-ocular fluids may be traced as follows: From the vitreous to the retina (fig. 3); between the cells of the retina to the choroid; through the lymph clefts of the nerve, particularly those about the central blood vessel and the sheath of the nerve (fig. 4), to the

6. Krug, Ernest F.: An Investigation of the Ferment Activity of the Rabbit's Vitreous, Tr. Am. Ophth. Soc., 1931, p. 451.

chiasm. From this point on, the pathway is the reverse of that just described, the termination of the path being the vitreous of the opposite eye. We have not considered the passage of the pigmented oils into the brain along another course of lymph channels, since this point is not in discussion.

In the present study no further histologic examinations were made, the observations being restricted to gross study of additional specimens. A group of twelve rabbits were given injections of 1 minim of a saturated solution of scharlach R in olive oil in the technic previously described. At intervals after the injection, varying from fifteen to sixty days, these animals were killed, and after proper fixation the eyes

Fig. 4.—Cross-section of an optic nerve with oil droplets in the sheath of the blood vessel and in the nerve.

were opened and the nerves and the chiasms examined grossly for deposits of pigmented oil. Photographs were taken of the gross specimens, and typical examples are appended herewith. This gross study was undertaken to demonstrate grossly that pigmented oil did pass from one eye to the other, the path such oil droplets pursued having been previously shown. Of this group of rabbits, all showed the migration of the oil toward the retina and back toward the nerve and into the nerve itself. Only one rabbit (rabbit 163) showed the stained oil in the vitreous of the eye into which injection had not been made.

### INDIA INK

A total of seventeen rabbits were given injections with india ink, (Higgins india ink, undiluted), the previously described technic being

used; 2 minims (0.12 cc.) of india ink was injected into the vitreous. These animals were killed at intervals varying from three hours to thirty-eight days after injection.

Fig. 5.—Wandering cell exudate covering the nerve head. Note that cells are all coming from one side, the side of injection.

Fig. 6.—Proliferation of the retina in encapsulation of injected india ink.

On ophthalmoscopic examination, the injected material was seen as a drop suspended in the vitreous at the point of injection. In twenty-four hours there occurred swelling of the iris. This slowly subsided, occasionally leaving irregularity and sluggishness of the pupil. The

iris became discolored (darker) and a change was noted in the color of the retina, which became either diffusely or in patches more or less grayish.

Histologic examination revealed the following changes: Within twelve hours after the injection the injected mass was more or less surrounded by a wandering cell exudate, and within twenty-four hours

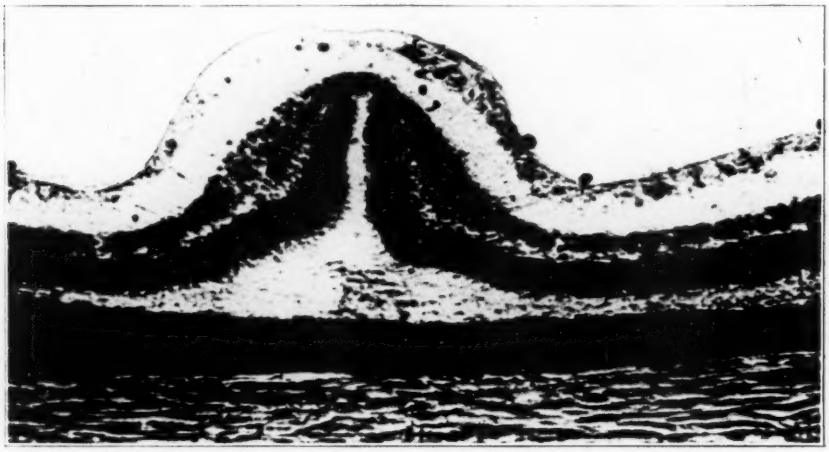

Fig. 7.—Distribution of india ink in the retina.

Fig. 8.—India ink in the retina.

the nerve head of the eye into which injection had been made was covered with a similar exudate (fig. 5). In forty-eight hours proliferative changes occurred, starting from the inner coat of the retina and proceeding to encapsulate (fig. 6) the injected material. This proliferative process was still active thirty-five days after injection. Beginning three days after injection, deposits of carbon were found scattered in diffuse fashion in the various layers of the retina, down to

Fig. 9.—Deposition of india ink in the retina.

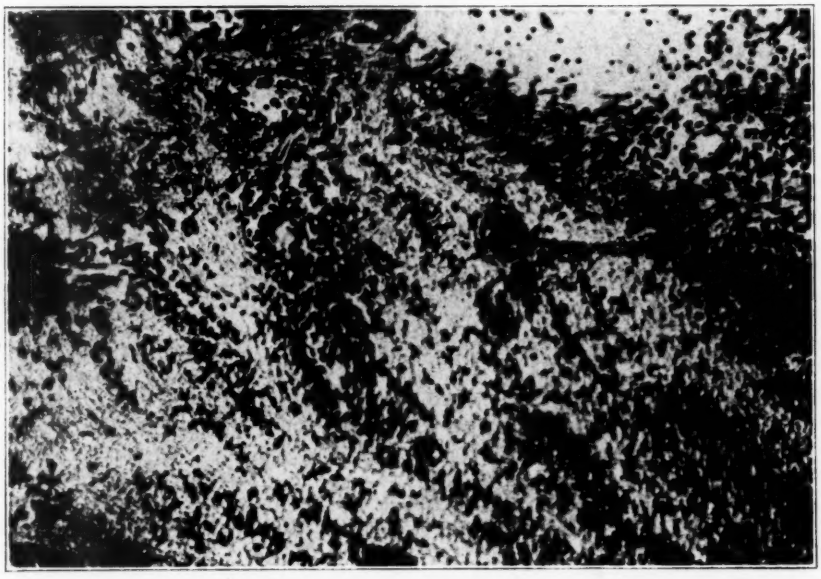

Fig. 10.—Round cell infiltration of the optic nerve. Note the grouping of the round cells about the lymph clefts.

the choroid. These deposits were thickest on the side of the eye on which the injection had been made (figs. 7, 8 and 9). At the same time, carbon granules could be demonstrated in some of the wandering cells which had surrounded the injected material. A wandering cell infiltration also was demonstrable in the nerve (fig. 10), with the point of maximum density in the sheath about the blood vessel. Many of these wandering cells contained carbon granules (fig. 11). After

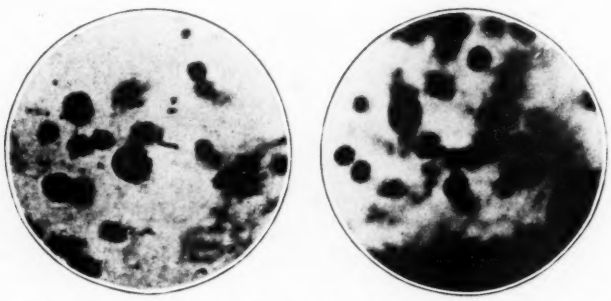

Fig. 11.—High power of cells in figure 10 showing carbon granules in cells.

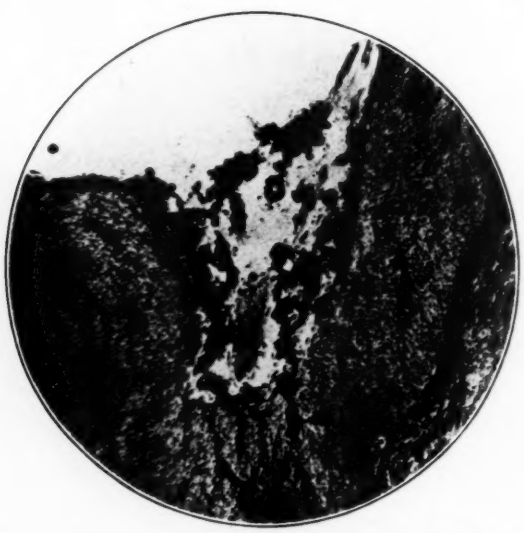

Fig. 12.—Carbon deposit in nerve head of the eye into which injection has been made. Note the distribution along the lymph clefts.

the seventh day, small granules of carbon were demonstrable in the nerve of the eye into which injection had been made (figs. 12, 13, 14 and 14 *A*), the chiasm, the nerve of the opposite eye and the nerve head of the eye into which injection had not been made (figs. 15 *A*, *B*, *C* and *D*). These deposits most probably had been made by wandering cells, which, having proceeded thus far on their journey, had died and had become autolyzed, leaving the pigment behind.

While these changes had occurred in the path from one eye to its fellow eye, another group of changes had taken place in the eye into which injection had been made. Beginning twenty-four hours after

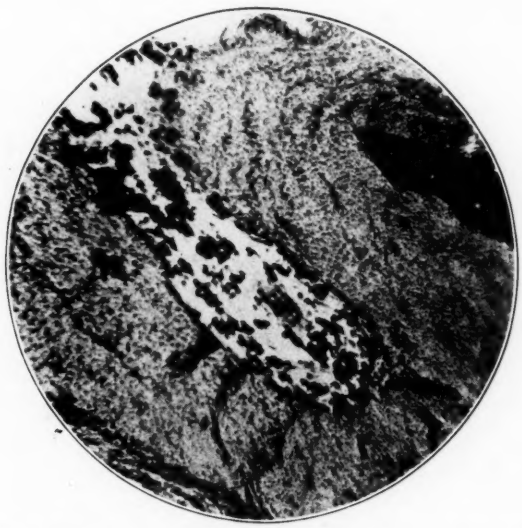

Fig. 13.—Carbon deposits in the nerve head of the eye into which injection has been made. Note the deposit along the lymph clefts.

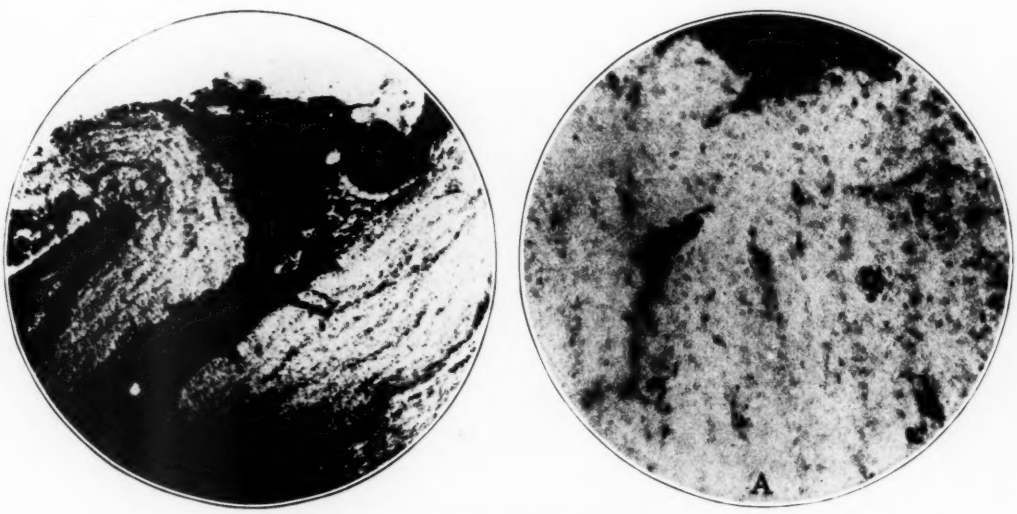


Fig. 14.—Carbon deposit in the nerve head of the eye into which injection has been made. Note the infiltration of the lymph clefts at the periphery of the column. *A* shows carbon in the lymph clefts of the nerve of the eye into which injection has been made.

injection, a wandering cell exudate occurred in the ciliary body (fig. 16). At subsequent intervals this wandering cell exudate became more intense, a sort of heaping up of the cells having occurred. The reac-

tion suggested that at this point there had been a blockade in the lymph system so that the wandering cells could not proceed further. A careful study of a number of normal rabbit eyes failed to demonstrate a dead end to the lymphatic system at this point. This, however, did not exclude the possibility that a dead end was present.

As might be expected after the injection of a foreign substance into the vitreous, an inflammatory (nonsuppurative) reaction occurred

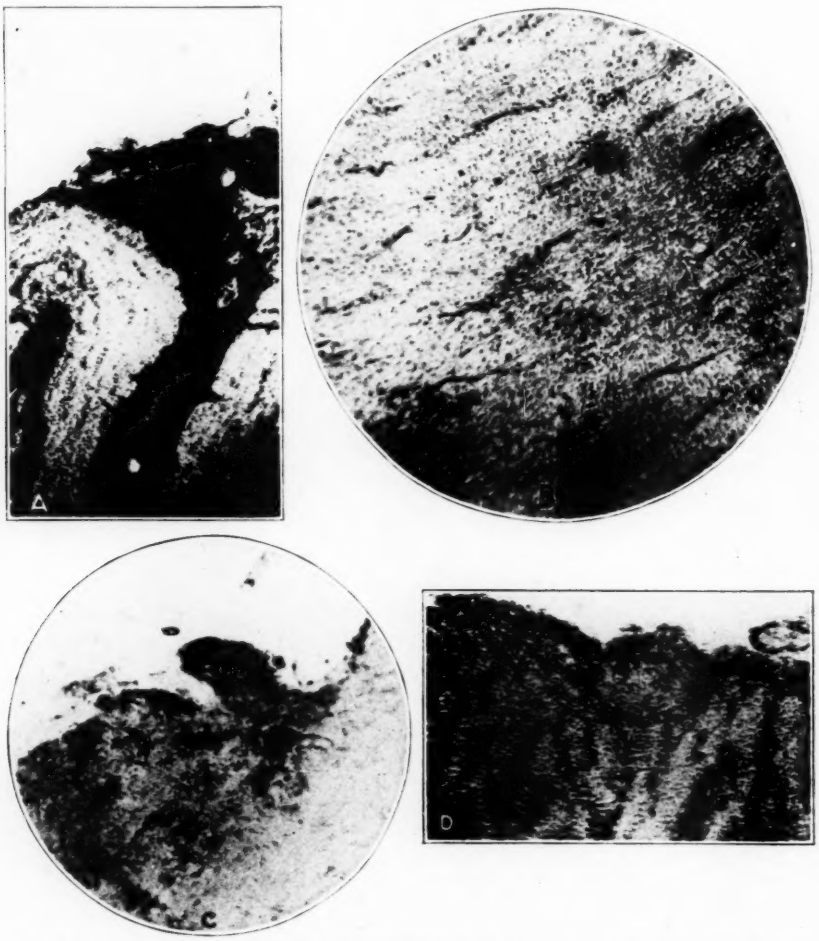


Fig. 15.—*A*, carbon deposit in the nerve head of eye into which injection has been made twelve days after injection. *B*, carbon deposits in the optic chiasm twelve days after injection of india ink. (Same animal as in *A*.) *C*, carbon deposits in the nerve head of eye into which injection had not been made twelve days after injection of india ink. (Same animal as in *A* and *B*.) *D*, carbon granules in the nerve head of the eye into which injection had not been made.

in the various coats of the eye. This reaction will not be described since it throws no light on the path which might be followed in the condition of sympathetic ophthalmia.

Fig. 16.—Cellular infiltration of the ciliary body.

Fig. 17.—Site of injection of kieselguhr twelve hours after injection.

KIESELGUHR

Fourteen rabbits were given injections in the vitreous with a suspension of kieselguhr in distilled water. This suspension was made by

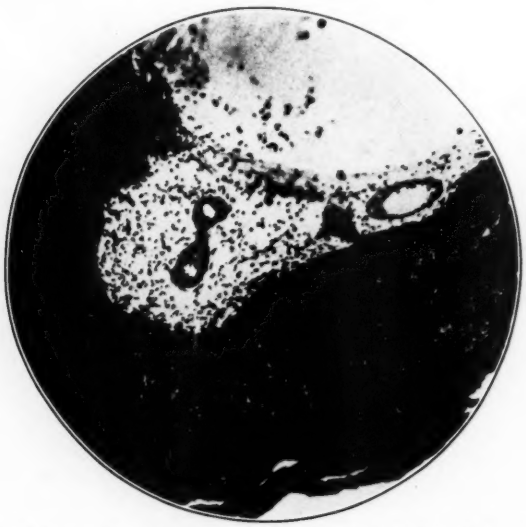

Fig. 18.—Wandering cell exudate in the nerve head after injection of kieselguhr.

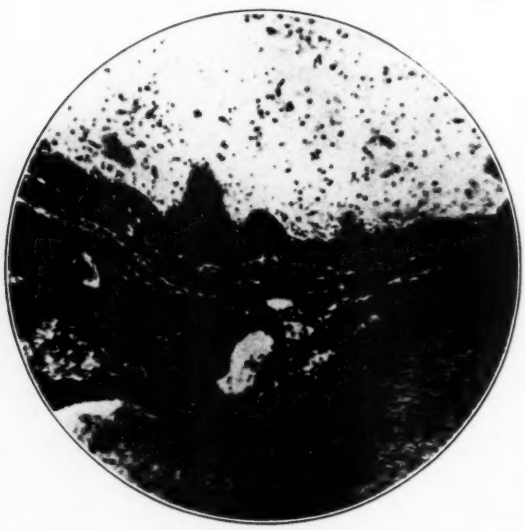

Fig. 19.—Inflammatory reaction of the ciliary body and iris and cellular infiltration of the vitreous after injection of kieselguhr.

placing 1 Gm. of kieselguhr in 100 cc. of distilled water, shaking vigorously for ten minutes, then placing the suspension at rest for one hour and using the supernatant fluid. The dose was 1 minim (0.06 cc.). The animals were killed at intervals of from three hours to one month.

Within twelve hours (fig. 17) after injection, a wandering cell exudate occurred about the point of injection. After twenty-four hours, this wandering cell exudate, coming down on the side of injection, began to infiltrate the nerve head (fig. 18). About this time a marked

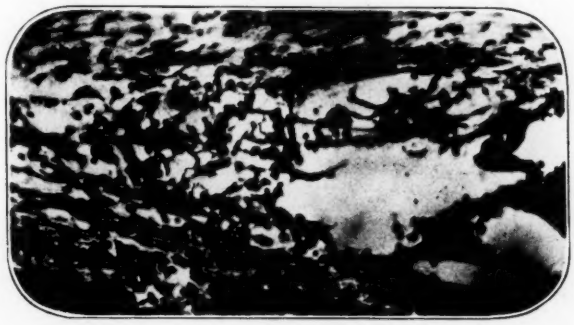

Fig. 20.—Inflammatory cell exudate in the ciliary body of the eye after intravenous injection of Streptococcus viridans.

Fig. 21.—Wandering cells in the vitreous just above the nerve head after intravenous injection of Streptococcus viridans.

inflammatory reaction of the iris with marked cellular infiltration of the vitreous also was observed (fig. 19). On the fifth day, wandering cells appeared at the nerve head of the eye into which injection had not been made. The path of the wandering cells was apparently the same as that described under india ink.

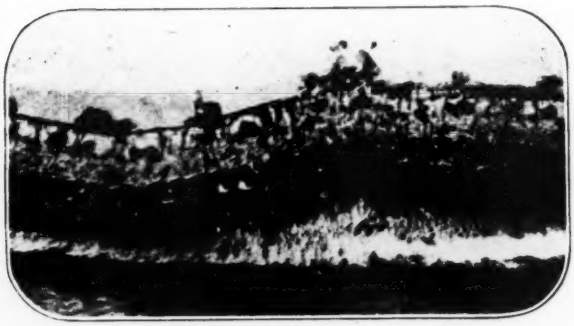

Fig. 22.—Retina of a rabbit given an intravenous injection of Streptococcus viridans. (Dalen plaques.)

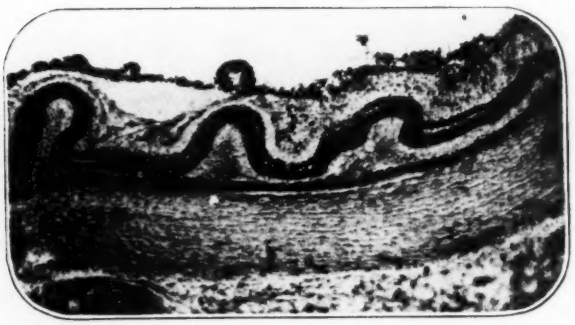

Fig. 23.—Exudate in the vitreous of an eye into which human tubercle bacilli was injected.

Fig. 24.—Exudate in the vitreous and wall of the eyeball after injection of streptococci.

LIVING BACTERIA

Five rabbits were given injections of living human tubercle bacilli in dosage of 1 cc. of a suspension standardized at one billion per cubic

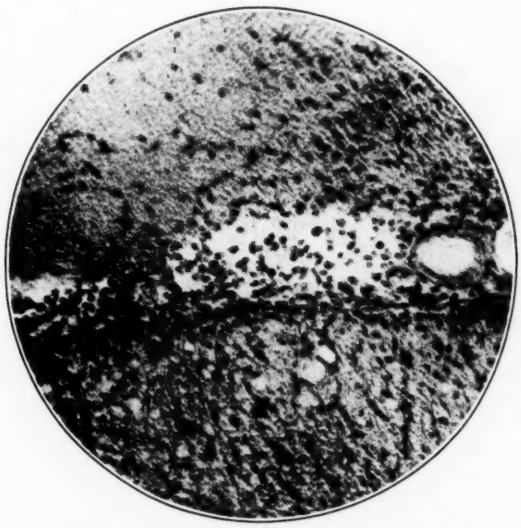

Fig. 25.—Plastic exudate covering the nerve head after injection of living bacteria.

Fig. 26.—Plastic exudate on the inner surface of the iris after the injection of tuberculin.

centimeter. These injections were given intravenously into an ear vein. In a similar manner, five rabbits were given intravenous injections with a suspension of Streptococcus viridans, the dose being 1 cc. and the suspension being standardized at one billion per cubic centi-

meter. These animals were killed at varying intervals up to three weeks after injection. No lesions were demonstrable in the eyes of the animals that had been given injections, except in one instance. In this animal, killed three weeks after intravenous injection of living Streptococcus viridans, an inflammatory cell infiltration was found in the ciliary body of one eye (fig. 20). Histologic examination showed that wandering cells were passing through the vitreous toward the nerve head (fig. 21). The retina showed no lesions except those clinically known as Dalén plaques—proliferation of the pigment epithelium of the retina (fig. 22).

Five additional rabbits received an injection of 1 minim of the tubercle bacillus suspension in the vitreous of one eye, and, in a final group,

Fig. 27.—Wandering cell exudate on the nerve head of eye into which injection had not been made; tuberculin was injected into the opposite eye.

five rabbits received similar injections of the suspension of Streptococcus viridans. These animals were killed at varying intervals up to three days after injection. The clinical appearance of the eye into which injection had been made was that of a suppurative panophthalmitis.

Histologically, both groups showed a cellular exudate in the vitreous. In the eyes of the rabbits into which tubercle bacillus had been injected, this exudate (fig. 23) was very much less than in the eyes of the rabbits into which streptococcus had been injected (fig. 24). In the early periods the exudate was restricted to the side of the eye on which the injection was made, but at a later period, it became diffuse throughout the vitreous. With this exudate came the usual group of inflammatory

reactions in the retina. The most suggestive observation made was that the nerve head in both groups of animals was covered more or less completely with a plastic and partially organized fibrinous exudate which closed the lymph clefts (fig. 25) and which acted as a mechanical barrier against the migration of the wandering cells. With the tubercle bacillus, the cellular exudate was restricted to the vitreous surface of the retina, and the deeper lying layers of this coat were not involved. With the streptococcus, however, this exudate involved all the coats of the eyeball. Again there was a recognizable heaping up of the inflammatory cells in the region of the ciliary body, suggesting a blockade, and here, as over the nerve head, there was an organizing plastic exudate.

It may therefore be concluded that living bacteria, when injected directly into the vitreous, produce such an inflammatory reaction that the lymph clefts become closed and migration along the channels previously described is not possible.

### TUBERCULIN

Fifteen rabbits received injections of 1 minim of old tuberculin into the vitreous. These animals were killed at intervals varying from one to thirty days after the injection. Initially, there was a pouring out of wandering cells, chiefly round cells, into the vitreous, which exudate was practically completely restricted to the side on which the injection was made. Some of these wandering cells proceeded to the inner aspect of the iris (fig. 26). Others gravitated to the nerve head through the vitreous. No wandering cells were demonstrable in the retina. The lymph clefts of the nerve were observed to be more or less filled with wandering cells and in one instance an exudate of wandering cells was observed on the nerve head of the eye into which injection had not been made (fig. 27).

It may also be concluded that a soluble foreign protein produces in the vitreous the same type of wandering cell exudate observed in other tissues of the body, after injection of similar substances. These wandering cells pass through the lymph clefts of the nerve head and proceed through the previously described channels to the nerve head of the opposite eye. A plastic exudate such as is observed when living bacteria are injected into the vitreous is not present.

### SUMMARY

The experiment demonstrates that particulate particles when injected into the vitreous can be carried from one eye to the other via the optic nerve and chiasm.

# Clinical Notes

## USE OF POTASSIUM HYDROXIDE STICKS IN THE GUIST OPERATION FOR DETACHMENT OF THE RETINA

BENJAMIN B. ADELMAN, M.D., NEWARK, N. J.

Recently the operation of Guist has come to the attention of ophthalmologists throughout the world as a promising treatment for detachment of the retina. This method consists in the cauterization with a stick of caustic potash of each trephine opening that may be made over an area of detached retina. Neutralization is made according to the method of Guist with acetic acid immediately after the cauterization. Potassium hydroxide dissolves collagenous tissue and brings about a plastic exudate. This latter causes adherence of the detached retina to the choroid.

Guist employs paraffin-covered sticks of caustic potash, which are tapered to a point. The paraffin tip is removed just a moment before the application to the exposed choroid. The sticks he has been using I find unsatisfactory for several reasons: (1) they are unwieldy; (2) they quickly become moist, as potassium hydroxide is hydroscopic; (3) when the stick is applied to the second or third trephine hole, it causes a much larger area of cauterization than is desired; (4) when the trephine area through the sclera is at or behind the equator of the globe, the stick cannot be applied properly, because of the proximity of other tissues, which may be damaged by the cautery; (5) the sticks are difficult to prepare.

At the New York Eye and Ear Infirmary, in Dr. Bernard Samuels' service, we have had occasion recently to perform the Guist operation in several cases of detached retina. I have substituted for the paraffin-covered sticks small glass rods, the tapered points of which are covered with potassium hydroxide. During the course of the operations, these rods were found to be light and easily handled, and they had sufficient potash to exert a proper caustic action. The rods may be discarded or may be washed, sterilized and prepared for further use. One rod is used for each trephine hole.

### METHOD OF PREPARATION

The rods are quickly made. Thin glass rods 7 cm. by 2 mm. are drawn to a point through a Bunsen flame. A stick of chemically pure potassium hydroxide is then melted down in a porcelain crucible until it is just liquefied. The tapered ends of the glass rods are then dipped in the liquefied potash and cooled. The dry caustic solidifies on the rod, and there is sufficient hydroxide on the tip to exert a definite effect. The rods are placed in test tubes, and kept in a safe, cool place. The time required for making these caustic tipped rods is roughly about ten minutes for a dozen. They are quickly and easily prepared in any hospital laboratory and can be kept indefinitely.

# OPERATING TABLE FOR ANIMAL EXPERIMENTS

H. F. Pierce, Ph.D., Baltimore

The simple and inexpensive operating table here described is in daily use in the research laboratories of the Wilmer Institute, and two years' experience has proved its convenience and its adaptability to a wide variety of animal experiments. Details of construction are given, since its manufacture is well within the capabilities of the usual university mechanic's shop.

The table is made of wood. It is 38 inches high, 55 inches long, and 21 inches wide. The legs are set back 3 inches from the edge of the table top. They are 3 inches square at the top, 2 inches square at the bottom, and cross-braced in each direction 6 inches from the floor.

The table top is 1¼ inches thick. On a line 1½ inches from the edge, parallel to it and running continuously around it, half inch holes are bored through the table top every 2 inches. The holes are chamfered at the ends so that they may be cleaned easily. These holes are for the purpose of mounting vertical steel rods, as shown in the figure.

The rods are of one-half inch steel (coppered Bessemer rod or drill rod, the latter being easier to machine, but more liable to rust), threaded for a length of 2¼ inches at one end with standard half inch machine thread, and fitted with a brass flange 2 inches in diameter and one-fourth thick. The flange is screwed tightly to the rod, and faced off in the lathe. The threaded end of the rod is inserted into one of the holes in the table top and fastened to it by means of a wing nut, which holds it rigidly in place but allows it to be removed in a moment. The rods may be made as long as desired, though the 18, 24, 30 and 36 inch lengths have been found to be most convenient.

With this table and the usual laboratory clamps, supplemented by a few 20 and 30 inch lengths of rod, it is a simple matter to erect a structure for holding an animal and mounting apparatus to suit the needs of any particular experiment. The rigidity of the structure is especially appreciated when a needle is to be held in the anterior chamber of the eye, or when delicate recording instruments are employed. For supporting a kymograph, an extension or temporary shelf may be fastened to the end of the table by means of short bolts fitted with wing nuts. Other uses of this equipment will suggest themselves to the operator, according to the needs of his research.

With dogs as the experimental animals, an animal board of simple slat construction is employed, as shown in the figure. It is 42 inches long, 12 inches wide and 5 inches high at its highest point and 3 inches at its lowest. It is fastened to the table by two one-fourth inch bolts fitted with wing nuts, and is easily removable for cleaning. Under it are two pans of stainless steel, each 18 by 12 by 1½ inches. Stainless steel withstands the action of blood, urine, salt solution and the usual laboratory chemicals, and is not liable to mechanical injury.

For rabbits and cats the metal animal holder made by the Harvard Apparatus Company is used, and is fastened to the table by a rod and

From the Wilmer Institute, Johns Hopkins University and Hospital.

clamps. The head holder for rabbits is of the Czermak type, and the dog head holder is of the Columbia pattern. Both are satisfactory. A word about clamps may be in order. The usual cast-iron laboratory clamps are adequate for joining rods and other rough work, but when accurate position and fine adjustment are necessary, precision clamps are essential. Those of Joseph Becker and Eberhard and Sons are satisfactory, and the universal precision clamp of Eberhard is particularly useful for holding a needle in the anterior chamber of the eye. Becker's fine adjustment clamp is a most convenient means of supporting and adjusting tambours, manometers or other instruments that write on a kymograph drum.

The operating table and dog board are finished with three coats of gray house paint. This finish is easy to apply and renew, and it has been found durable and resistant to ether, alcohol and the usual laboratory chemicals.

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## A CASE OF CYANOSIS DEMONSTRATING THE CIRCUMCORNEAL VEINS

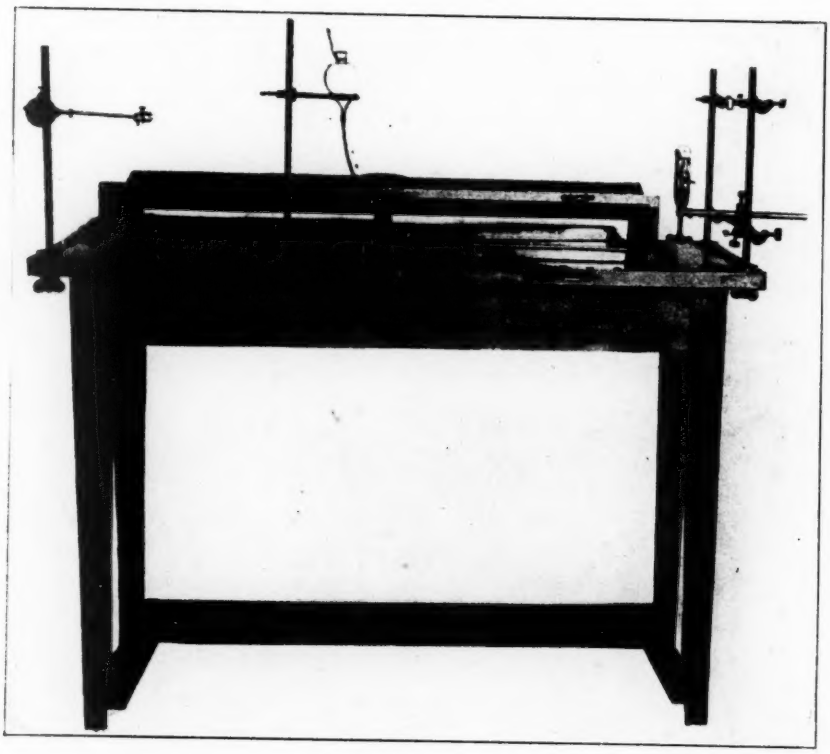
ROBERT BLUE, M.D., CHICAGO

As a teacher of ophthalmology to undergraduate students, I have always laid great stress on the recognition and clinical interpretation of circumcorneal injection. I am impelled, therefore, to present the appended case of engorgement of the circumcorneal vessels, which shows in a remarkable manner their anatomic distribution.

Ordinarily it is the anterior ciliary arteries and their peripheral terminations, the circumcorneal loops of vessels, that are engorged. This is the case in all acute inflammatory conditions affecting the cornea, iris or ciliary body. In the case presented, the anterior ciliary veins were engorged, but as in this location the veins accompany the arteries, the study from an anatomic standpoint is not vitiated. In fact, it may be that it is enhanced, for in this instance the circumcorneal loops of vessels that spring from the anterior ciliary arteries just before they penetrate the sclera were not shown, but the emergence of the veins from the interior of the eye was shown.

These small veins as they emerge from the eye continually unite, forming an anatomic ring surrounding the cornea. The anterior border of this ring lies from 1 to 2 mm. behind the corneoscleral margin. The ring of sclera lying between the points of emergence of the anterior ciliary veins and the peripheral margin of the cornea is white and devoid of congested vessels. It is precisely this scleral ring that is supplied by the circumcorneal loops of vessels that spring from the anterior ciliary arteries just before they plunge through the sclera to empty their contents into the circulus arteriosus iridis major. And it is precisely

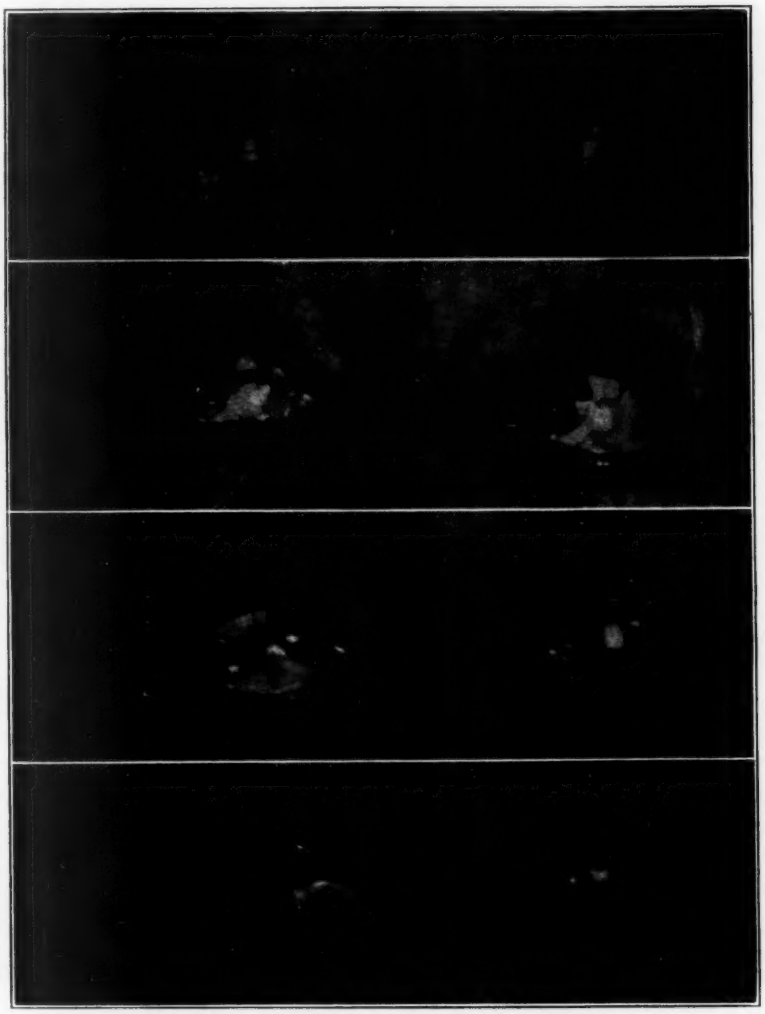
From the Department of Ophthalmology, Northwestern University Medical School.



Operating Table.

this scleral ring bordering the cornea which is most intensely red in the typical circumcorneal injection accompanying inflammatory lesions of the cornea, iris or ciliary body.

Why are not these peripheral vessels—the circumcorneal loops of vessels—engorged? I assume that it is because they are fed by the



The patient's eyes, showing the anastomotic ring of veins.

anterior ciliary arteries and are hence primarily arteries, while the vessels engorged in the case presented were veins.

It is proper, then, to speak and to think of the circumcorneal loops of vessels as arterial loops, although in all schematic drawings of the circulation of the eye the return members of the loops are pictured as veins. If one chooses to look at the facts in this way, it is proper to say that the anterior ciliary arteries, just before plunging through the sclera, give off small arteries that approach the peripheral border of the

cornea and there loop back on themselves and empty into the anterior ciliary veins or the anastomotic ring of veins that lies from 1 to 2 mm. behind the cornea. It is this anastomotic ring of veins that is so clearly shown in the accompanying picture.

It is customary, furthermore, to speak of the circumcorneal loops of vessels as springing from the arteries just before they plunge through the sclera. In this connection it is well to bear in mind the fact that these arteries also form an anastomotic ring comparable to the venous ring so clearly shown. It is interesting to note further that the ophthalmoscope reveals the retinal veins similarly engorged.

Dr. P. A. Sullivan supplied me with a brief résumé of the general clinical findings. The case was one of mitral insufficiency accompanied by edema of the lower limbs and shortness of breath on slight exertion. There had been periods when the break in the compensation of the heart had been sufficiently severe to cause grave symptoms. The case is comparable to the rare cases of patent foramen ovale in which the general cyanosis is made more striking by the appearance of the eyes, the circumcorneal vein being unduly prominent.

It is not the purpose of this communication, however, to deal with the medical aspects of the case, but more especially to call attention to the anatomic characteristics that were so pronounced that they show fairly well in the accompanying photographs, which, in order to preserve accuracy, have not been retouched.

The photograph was taken by Miss Madge Walsh, photographic department of Northwestern University Medical School.

# Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

## PATHOGENESIS OF SENILE CATARACT

DANIEL B. KIRBY, M.D.
NEW YORK

Cataract may be defined as any opacity of the crystalline lens. For the scope of this review senile cataract may be considered as any opacity arising in the decline of life, that is, after the adult period has been reached or passed. Duke-Elder quoted Horlacher, who found the changes of senile cataract in 65 per cent of persons between 51 and 60 years, and in 95 per cent of those above 60 (Barth, quoted by Duke-Elder). It arises at an earlier period in certain of the oriental (Kirkpatrick) races than it does in the occidental races.

The literature on the pathogenesis of cataract is so voluminous that only significant and representative articles will be quoted or analyzed. Necessarily more space will be devoted to recent thought, as the older ideas have been disproved or cast aside because they give no aid to progress in the understanding of this complex biophysicochemical problem.

Hippocrates (quoted by Siegrist) was the first writer and physician who mentioned a bluish color to the lens, which he probably saw in a case of senile cataract. Cornelius Celsus (5 B. C.) believed that some "perspired matter caused a suffusion" and appeared as turbidity. According to Galen, "hypochyma" was formed by a "falling down" of coagulated fluid back of the iris. This concept of cataract was still authoritative in the middle ages, when the term cataract was employed. Treatment was by couching or depression of this material back into the vitreous.

Modern research on cataract began in 1643, when Quarre taught that cataract was not coagulated fluid, but a turbidity of the lens. Remigius Lasnier was the first surgeon who claimed that in treatment he did not push back a membrane, but the lens itself. Rollfinck proved that anatomically cataract is a disease of the lens, and in this he con-

From the Department of Ophthalmology, College of Physicians and Surgeons, Columbia University.

This article was prepared during my Research Fellowship in Ophthalmology for the American Academy of Ophthalmology and Oto-Laryngology.

firmed Quarre's theory. Pierre Borel (1653) and Peter Cassendi (1592-1655) substantiated his claim. Nevertheless, most of his contemporary colleagues upheld Galen's ideas. A few years later the new ideas again had been forgotten, until two French physicians, Brisseau and Antoine Maitre-Jan, succeeded in proving, after long disputes, that Galen's school was wrong. Others upheld the new theory, and in 1708 the Parisian Academy approved of it officially.

## POSSIBLE REACTIONS OF LENS CELLS

To begin with the modern conceptions it is well to bear in mind the thoughts of Friedenwald on the possible reactions of the cells of the lens:

1. The cells may die slowly and become sclerosed. This sclerosis of the central parts of the lens proceeds normally with age, and unless it is extreme it impairs the transparency of the lens very little, though it causes a change in refraction. In extreme instances the transparency of the lens is markedly impaired, because the sclerosis proceeds unevenly and leads to the development of multiple, irregular refracting surfaces within the lens, the sclerosed lens substance having a higher index of refraction than the living fibers. The normal lens is distinctly less transparent than is water or glass to waves of ultraviolet rays. When the fibers become sclerosed, this tendency to absorb the shorter wavelengths becomes exaggerated and a definite encroachment on the visible spectrum results. The sclerosis of the nucleus of the lens may proceed beyond the point that has come to be considered as normal; the encroachment on the visible spectrum becomes more marked, so that gradually not only the violet, but also the blue and finally the green and even part of the yellow rays become strongly absorbed. Correspondingly, the lens, which in childhood is colorless and in normal old age is slightly yellowish, becomes deeper yellow, then orange, then red and finally a dusky brown.

2. When the lens fibers die more rapidly they become autolyzed, and from the resulting fluid various constituents are precipitated, either amorphously or in the form of crystals. The mixture of precipitates and fragments of as yet unaltered lens fibers suspended in a fluid of high protein content transmits light very poorly. Later the individual fibers break up into chains of droplets (Morgagnian globules) of varying size. At about this stage the lens often swells slightly. As the fragments of lens fibers begin to be digested, the intercellular fluid changes from a watery to an albuminous one. The process is generally slow, involving a few fibers at a time. Eventually the diffusible products of tissue disintegration escape through the capsule, and as the dead tissue is removed, the capsule shrinks and wrinkles.

3. The only cells in the lens capable of regenerative activity are those of the subcapsular epithelium. In response to mild injury these cells proliferate, sometimes forming a dense opaque membrane many cells deep. In response to certain injuries of the posterior cortex, the capsular epithelium may proliferate backward until a continuous layer of epithelium is formed, surrounding the whole lens just beneath the capsule.

These three reactions, together with the calcification and lipoid infiltration of deal tissue, form the whole of the pathologic possibilities of the lens. All of them lead eventually to optical imperfections in the lens.

## LOCATION OF OPACITIES OF THE LENS

Magnus classified cataract as beginning at the equator of the lens in 83 per cent, at the poles in 9 per cent and in the nucleus in 8 per cent of cases.

Knapp found that senile cataract commonly presented the same character in both eyes at the onset, when three principal forms can be distinguished: (1) a form commencing circularly with points or striae, usually radiating, in the equatorial region, which may be subcapsular or supranuclear (about one third of all cases); (2) a form beginning in the lower half of the lens, usually to the nasal side, most often a crescentic opacity parallel to the equator in the posterior cortex about 1 or 2 mm. from the margin of the lens, rather less often as a large radiating wedge (about one half of all cases); (3) a nuclear form beginning in 1.5 per cent of the cases; (4) a form commencing as a posterior cortical type in 6.3 per cent.

In 1927 I found the following types of senile cataract with the aid of the slit-lamp microscope: Of eight hundred and fifty-six cases of cataract examined, 52 per cent were of the cortical variety, 29 per cent of pathologic nuclear sclerosis and 3.5 per cent posterior cortical, while the remainder were combined, 9 per cent nuclear and cortical, 4 per cent nuclear and posterior-cortical and 2.5 per cent cortical and posterior-cortical.

## THE INFLUENCE OF HEREDITY AND ABIOTROPHY

Vogt expressed the belief that the duration of life is predestined by factors of inheritance. Just as graying or loss of the hair and wrinkles of the skin cannot naturally be prevented, because they are symptoms of old age, so is cataract a symptom of old age. All parts of the eye are bound to undergo senile changes. It would be strange if the lens should make an exception.

Löwenstein also considered cataract physiologic after 50 years of age. However, Burdon-Cooper expressed the opinion that it is incorrect to assume that cataract is nothing more than a sign that the predeter-

mined life of the lens cells ends sooner in some people than in others. In other words, opacities of the lens do not occur as a result of a process of abiotrophy. Burdon-Cooper associated them with the regressive changes in the metabolism of old age. He also expressed the belief that there is undoubtedly a hereditary influence in these factors. Parsons (also Hess and Sauermann), however, stated that there is no positive evidence regarding simple senile nuclear or cortical cataracts. Most cataracts that have been traced through family trees have shown a tendency to anticipate the time of development somewhat in succeeding generations and to appear earlier, so that one may imagine them eventually getting to the child in utero to produce congenital cataract. These types of hereditary cataract are, however, not of the ordinary senile varieties, but run to the fusiform, axial, spindle (Knies, Leber), corralliform (Gunn), lamellar or zonular (Nettleship) type.

## MODERN THEORIES

Parsons pointed out in 1906 that the chief modern theories of the pathogenesis of cataract may be grouped into three classes: (1) that in which the cause is to be found within the lens itself; (2) a chemico-physical theory in which the nutrition of the lens suffers from the nature of the pabulum supplied to it by the ciliary body, and (3) that in which the cause is to be discovered in some general disease or a metabolic and nutritional disturbance.

There may be added, in a special class, the hypothesis that attributes senile cataract to the action of specific cytotoxins.

### I. THE CAUSE IS TO BE FOUND WITHIN THE LENS ITSELF

*Excessive Efforts of Accommodation.*—Schoen (quoted by Siegrist) attributed cataract to increased efforts of accommodation which caused folds and tears in the capsule between the zonular filaments, producing swelling of the epithelium. Hess (quoted by Burdon-Cooper), while upholding the Helmholtz theory of accommodation, felt that the opacity might result from slits produced between the zonular filaments by accommodative pressure against a nonyielding nucleus. According to Burdon-Cooper, hypermetropia has a much greater influence on the development of opacities of the lens than myopia (excluding degenerative myopia), owing to the greater accommodation necessary to its correction and the adverse influence on the ligament and capsule. He stated that in a great proportion oblique astigmatism is more potent in this respect because in these there is an irregular torsional accommodation effort, which produces an irregularity in the shape of the lens.

*Irregular Sclerosis of the Nucleus of the Lens.*—Elschnig has found a disproportion between the shrinking of the nucleus and the regenera-

tive activity of the epithelium due to the reduced vitality of old age. The lens is soft in youth, but the formation of a hard nucleus becomes true at about the twentieth year. This nucleus, with progressing years, becomes denser and smaller. In compensation for its lost volume with the shrinking of the nucleus, the epithelium of the lens regenerates. Burdon-Cooper also postulated an irregular sclerosis of the nucleus of the lens, and stated that this impedes the flow of nutrient materials, but asked why this occurs only in some lenses and not in all. Becker (quoted by Siegrist) suggested an anomalous chemical behavior of the nucleus to account for it, and considered that the chemical changes in the nucleus preceded by years those in the cortex. Deutschmann (quoted by Magnus) suggested that the water given up in the sclerosing of the nucleus clouded the lens by swelling its cortical fibers.

*Changes in the Permeability of the Capsule of the Lens.*—The capsule of the lens acts as an envelop, protecting the lens in much the same way as the envelop prevents the escape of hemoglobin and limits the entrance of water into the red blood corpuscle. Kirby (1931) has drawn attention to the similarity existing between certain elements in the lens and in the red blood corpuscle.

The capsule and epithelium of the lens guard it. Some object to this function of the epithelium, because the latter does not line the posterior capsule. But here the lens fibers are inserted by broad ends, forming a fine mosaic that acts like epithelium. Fluid from the vitreous enters the lens only with difficulty; hence a wound in the posterior capsule results in little opacity.

Friedenwald stated that perhaps the closest approach to an understanding of the local mechanism responsible for these changes in the lens is to be found along the lines of some of his recent investigations. The lens depends for its nutrition on the permeability of its capsule, through which foodstuffs and metabolic products dialyze in and out. It can be shown that the permeability of the capsule regularly decreases with age, and, further, that a decrease in permeability that is within the range of physiologic experimentation is sufficient to initiate the development of cataracts. The hypothesis can, therefore, be advanced that some forms, at least, of senile cataract result from an exaggeration of the normal senile decrease in capsular permeability.

Burdon-Cooper studied the surface tension of the aqueous humor in cases of senile cataract and found it high, approximating more that of water than the normal. There is a more rapid interchange between the aqueous and the lens in cataract than in the normal eye with the abstraction of the soluble albumin and products of the cataractous change. He also stated that in the senile subcapsular variety, the opacity starts in the outermost cortical layers, giving rise to the appearance

of cracks or fissures, which later form dark radial sectors. These and subcapsular vacuoles start by an increased permeability.

Duke-Elder stated that an increase or a derangement in the permeability of the capsule of the lens, effected either by the incidence of radiant energy or by metabolic or traumatic influences, allows the abnormal entrance of fluid or electrolytes into the lens, which may readily lead to the coagulation of its protein. On the other hand, this may facilitate the escape from the lens of some constituents, such as glutathione, which are essential for its metabolic activity.

*Changes in the Epithelium.*—Schoen (quoted by Siegrist) stated that inflammation and abuse of the eyes may lead to swelling of the epithelium of the lens which will disturb its nutrition. Proliferation of the epithelium has been demonstrated in many cases (Parsons, Elschnig). The epithelium has undoubtedly an important function in controlling the metabolism of the lens, besides providing new cells for the cortex of the lens, which produces the lens fibers. Kirby (1926) has grown epithelium of the lens in pure cultures, and Adachi has proved that this epithelium grown in vitro produces the typical organ-specific lens protein. Hess rather expressed the belief that a general disturbance of the whole organism causes cataract by producing the slow death of the lens epithelium. When the epithelium decays, its protective action is missed and excess fluid may be absorbed.

*Changes in the Protein of the Lens and Its Fractions.*—Burky and Woods (1927) have given an interesting review of this problem. In 1894, Mörner isolated from beef lens two crystalline fractions, which he called "lens alpha" and "lens beta," and an insoluble substance, which he termed "albuminoid." In 1910 and again in 1922, Jess reported that in old age and in cataractous lenses the amount of alpha fraction was decreased. In 1903, Uhlenhuth reported his discovery concerning the organ-specific immunologic properties of protein of the lens. Hektoen showed that the alpha and beta crystallins of the bovine lens were precipitogenically distinct. Burky and Woods have discovered a third protein, gamma crystallin.

As early as 1904, the observations of Uhlenhuth concerning the possibility of a relationship between protein of the lens and lenticular disease excited interest among clinicians and investigators. In 1902, Davis began working on this problem, publishing his first paper on this subject in 1922. As a result of Davis' clinical observations, he was convinced that immunization with a preparation of the bovine lens has a definite effect on retarding the development of lenticular opacities. The same subject was investigated experimentally by Paul Römer. Although he could not substantiate his hypothesis by experimental research, he was apparently convinced that the formation of cataract

was due to a reaction taking place between the blood serum of the patient and the protein of the lens. The experiments of Guyer and Smith, although not primarily concerned with the immunologic reactions of protein of the lens, took advantage of the fact that in embryonic life the lens is surrounded by the tunica vasculosa and is therefore more subject to influences from blood serum than would occur later in life. These observers immunized fowls to the protein of the lenses of rabbits, producing anti-rabbit lens serum. This serum was then reinjected into pregnant rabbits at intervals of two or three days over a period of two weeks. Among sixty-one young rabbits that survived, nine showed ocular defects centered chiefly around the crystalline lens, imperfect development or liquefaction of the lens, partial or complete cataract or other ocular lesions, which, in the opinion of these authors, were due primarily to the imperfect development of the lens. Poynter and Allen have not been able to verify these observations, and they expressed the opinion that a peculiarity in the strains of animals used by Guyer and Smith had much to do with the anomalies produced.

*Allergy of the Protein of the Lens.*—Römer administered lens extract by mouth and Davis by subcutaneous injection as a therapeutic agent in the treatment of cataract. In 1922, Verhoeff and Lemoine described endophthalmitis phaco-anaphylactica, and announced the indications for treatment with protein of the lens for this specific condition. Their conclusions were later sharply challenged by Röth, and these objections were later replied to by Verhoeff. Berens and his co-workers reported observations on several patients with hypersensitivity to lens extract who were treated by injections of lens extract, both as a prophylactic before operation and as a therapeutic agent in combating postoperative inflammation after extraction of the cataract. These attempts at therapeusis with lens extract have not been based on the determination of a specific allergic state, except in the specific cases reported by Verhoeff and by Berens and his associates.

Burky and Woods (1931) found positive allergic cutaneous reaction to protein of the lens in 17 per cent of eighty-six patients tested. The majority of these positive reactions were associated with postoperative inflammation after extraction of the cataract (in persons over 60 years of age).

Thus far the relationship between a specific allergic state and the development of senile cataract has not been established.

*Spontaneous Precipitation of Beta Crystallin.*—In a mixture of alpha and beta crystallins, the beta protein in the mixture does not show any tendency to spontaneous precipitation in the higher ranges from $p_H$ 7.8 to $p_H$ 6.2, whereas it does tend to precipitate when in a pure condition.

Apparently the presence of the alpha fraction has an inhibitory effect on spontaneous precipitation of the beta fraction between $p_H$ 7.8 and $p_H$ 6.2.

*Wood's Theory Involving the Precipitation of Beta Crystallin.*—The tendency of beta crystallin to precipitate spontaneously is of great interest, as it offers a plausible hypothesis to explain the etiology of senile cataract. The gradual decrease in the alpha fraction allows the beta fraction to become relatively greater and purer, and further gradually removes the protective colloid action against its spontaneous precipitation. Spontaneous precipitation of beta in vivo and in situ may now occur, with resultant clouding of the lens. This is an especially interesting hypothesis when it is considered that the range of spontaneous precipitation of the beta fraction lies in the normal hydrogen ion concentration of the body fluids. Such a theory is, however, as Woods stated, totally without experimental proof in vivo.

## METABOLISM OF THE LENS

*Autoxidation System.*—For growth and for maintenance of transparency of the lens, an active metabolism that is chiefly concerned with oxidation and reduction is necessary. In the body there are two principal methods of exchange: one by which oxygen and carbon dioxide are given off or taken up by the blood to and from the tissues, and the other according to the autoxidation system. Loeb Cascio (quoted by Duke-Elder) claimed to have demonstrated the presence of oxidases and catalases in the lens, but Duke-Elder expressed the belief that these may have only a secondary activity in the oxidation of certain fatty acids (Ahlgren), while Goldschmidt (1924) and Adams (1925) have demonstrated that the internal metabolism of the lens is chiefly carried on by an autoxidation system wherein glutathione acts as a hydrogen donater while beta crystallin acts as a thermostabile residue. Oxidation is effected by removing hydrogen, and reduction is accomplished by supplying hydrogen. As the beta crystallin decreases with age, this system decreases in efficiency. Beta crystallin and glutathione are both diminished or absent in the lens in the presence of cataract.

*Derangement of the Autoxidation System.*—The cause of cataract may be a derangement of the autoxidation system of the lens. This, as Duke-Elder stated, may be caused by the direct action of radiant energy on the lens itself, decreasing its glutathione content, or on the capsule, the efficiency of which is thus lowered. Alternatively, the autoxidation system may be deranged by alterations in the concentration of hydrogen ions and salts induced by the factors already mentioned.

*Changes in the Protein of the Lens.*—Duke-Elder stated that protein may be changed from the colloidal to the particulate form by two distinct processes, precipitation and coagulation. 1. The first process,

which is brought about by the action of concentrated salts such as ammonium sulphate, is a reversible and purely chemical change whereby aggregates of chemically unchanged particles are thrown out of colloidal solution in a condition capable of reversion to their original colloidal state. 2. The second is a complex process which takes place in two stages: (*a*) denaturation whereby the natural protein is chemically altered to a form in which it is readily coagulable; (*b*) agglutination, a physical process whereby coagula are formed by flocculation of the altered particles.

Scalinci (quoted by Pisani) has evolved a theory based on the chemical properties of the lens. He considers the lens a mass of organic hydrogel, which he calls phacoprotein, one part of which is soluble while the other is insoluble. These normally are alkaline. Faint organic acids, such as acetic, oxybutyric and formic acids, may find their way to the aqueous, and by neutralizing the phacoprotein they form precipitates in the lens of neutral phacoprotein.

*Hydration of the Protein of the Lens.*—Deutschmann (quoted by Siegrist) expressed the belief that opacity of the lens is caused by the absorption or "taking up" of liquid by the lens fibers, which swell and in this way are destroyed.

Dor's (quoted by Magnus) theory of hydration was based on: 1. Deutschmann's analysis, which showed that cataract contained more water than normal. 2. In cortical cataract the nucleus gains and the cortex loses in density, and protein escapes. 3. The percentage of soluble protein is less in cataract than in the normal lens, and sometimes disappears entirely from the nucleus. 4. The electrical resistance of the aqueous is diminished.

*Hydrolysis of the Protein of the Lens.*—Burdon-Cooper has rarely found a cataract bulkier or heavier than the normal lens. He felt that Dor really meant hydrolysis, which is simply a decomposition resulting from the assimilation of a molecule of water by the protein molecule of the lens with the production of entirely new substances. It was the detection of tyrosine, one of the cleavage products of protein, that led Burdon-Cooper to the recognition of the nature of the change, and he has no doubt that the fat or fatty acid, also present in cataract, will, when identified, confirm his belief. The theory accounts for the disappearance of the soluble protein of the lens, which is hydrolized and carried away.

*Color of Cataract.*—Sclerosis consists in a loss of soluble protein or its conversion into insoluble albuminoid. With increasing sclerosis, the nuclear portion becomes colored, from light amber to brownish red. In extreme cases, clinically known as black cataract, no opacity is present, the defective vision being due to absorption and reflection of the light.

Spectroscopic examinations by Hess and others have failed to detect the presence of any blood pigment. Burdon-Cooper found that the color is due to the oxidation of tyrosine, the amino-acid being split off from the protein molecule by a process of slow hydrolysis and then immediately oxidized by a ferment.

*The Effect of Radiant Energy on the Lens.*—Duke-Elder stated that the fundamental cause of cataract in all its forms, with certain exceptions, may be traced to the incidence of radiant energy directly on the lens itself. It was with the absorbed portions of the incident energy that he was particularly concerned in a consideration of the possible pathologic effects on the lens:

1. The longer wavelengths produce a thermal effect on the capsule and on the capsule and epithelial cells with tissue death by coagulation of the protein. The shorter wavelengths produce a typical abiotic effect and derange the permeability of the capsule, faciliating the interchange of water and salts.

2. On lens substance, radiant energy produces an opacity, or so modifies the colloid system that opacity is more easily produced by other substances.

The mechanism of the formation of opacity by radiant energy may therefore be ascribed to: (1) the entrance of fluid in a quantity sufficient to form vacuoles between the lens fibers; (2) coagulation of the protein of the lens, which results from one or another or all of the following factors: (*a*) mechanical strain on the fibers inducing denaturation, comparable to the effect on the surface of a soap bubble or at the interface between two immiscible liquids, (*b*) a change in concentration of salt, (*c*) a change in hydrogen ion concentration and (*d*) diffusion outward through the "now" permeable capsule of the important elements in the autoxidation system—the glutathione and beta crystallin.

Duke-Elder expressed the opinion that the work of Burge showing the marked influence of calcium salts in the coagulation of the protein of the lens by ultraviolet rays is to be associated with the cause of cataract in such conditions as tetany and myotonia.

Electric cataract was produced by Hess (1888) and Kiribuchi (1900) in animals by a discharge from the Leyden jar. The effect was most probably produced by electric oscillation or concussion.

Infra-Red Waves: Isolated heat rays readily produce opacity of the lens. Concentrated light from the carbon arc produces a disklike opacity on the anterior surface of the lens in albino rabbits in five minutes. Less intense exposures produce a diffuse opacity, and still less intense exposures, a postcortical cataract after some days.

Visible Rays: Opacity has been produced by concentrated visible sunlight (ultraviolet and infra-red excluded).

Ultraviolet Rays: Intense irradiation has produced opacities. Opacities of lesser degrees are produced if the exposed lens is then immersed in salt solutions.

Short Waves: Radium has produced cataract in animals, in the fetus, in utero and in man. X-rays have the same effect.

*The Effect of Heat Waves.*—Cataract is readily produced by heat acting directly on the lens. If the pigment of the eye that absorbs heat can be removed, as it was in the experiments conducted by Vogt in the albino rabbit, cataract is readily produced. The cornea does not always turn white, because the heat may be focused in the lens and not in the cornea. The latter tends to repair better. The direct action of the heat is possibly the principal factor in coagulation of the protein of the lens, but there is probably secondarily an increased permeability of the capsule, which permits abnormal fluid traffic through the capsule.

Molten glass gives ultraviolet only down to 3,340 angstrom units. while the infra-red rays are extremely powerful. The crystalline lens absorbs rays up to 4,000 angstrom units. Glass workers are subject to exposures lasting with intermission for years, bending over a tank and looking at a source of maximum intensity. The effect is most probably due to the ultraviolet rays. It is certainly not all due to the infra-red rays. There may also be a factor of malnutrition, owing to changes in the composition of the aqueous and to the effect of heat on the iris and ciliary body (Parsons). The cataract is usually complicated, owing to the mild uveitis that is present.

*Cataract in India.*—The conditions that may account for the greater prevalence of cataract in India may here be considered.

Pisani has written his thesis on:

Distribution: Cataract is much more prevalent in the northern and central division of India than in the lower provinces and in Burma, judged by the statistics of the number of operations performed annually.

Climate: There are three seasons, a hot, a rainy and a cold season. In northern India, the winter is colder and the hot season hotter than in southern India from April to June. Records from the Punjab, the northwest frontier and the United and Central provinces show the highest figures for temperature and the lowest figures for humidity for all India.

Diet: It has been stated that cataract is less prevalent among those who eat rice than among those who eat wheat and other similar grains, but the general opinion of medical officers in India is that diet has no connection with cataract. The well-to-do suffer less than the poor. This fact has linked up the factor of malnutrition as the cause in the latter, but the same conditions of poverty are found all over India. This cannot account for it in view of the geographical distribution.

The Influence of Intense Light and Glare: The light in India is intense, not only directly from the sun, but also reflected from the dry ground, which in the hot season is devoid of vegetation to a great extent. However, Pisani was convinced that light is not the direct cause.

The Influence of Heat: Heat may act directly on the eyes, or it may act indirectly by modifying the composition of the aqueous. During the hot season, the temperature in the shade rises in India to 120 F., and directly in the sun's rays, to from 160 to 165 F. The relative humidity may be as low as from 40 to 15. At times, especially in the early part of the hot weather, westerly winds are prevalent and are almost unbearable because of the high temperatures. As an instance of the great heat, Pisani mentioned that he was never able to wear dark glasses because the glass became so hot as to produce an intensely uncomfortable action of dryness of the eyes. It can readily be imagined that the conditions that prevail in upper India must produce an intense local action on the eyes, and indirectly on the composition of the aqueous by the excessive loss of fluid from the system. However frequently this loss may be replaced, there must be derangements, and these, even if temporary, may, when frequently repeated, modify the composition of the aqueous. Outside the body the protein of the lens coagulates at a temperature of 52 C. (149 F.).

## II. THE CHEMICOPHYSICAL THEORIES

The nutrition or metabolism of the lens may be disturbed because of the change in quality or quantity of the pabulum furnished in the aqueous.

This necessarily involves consideration of the method of formation (Yudkin) of the aqueous. It is granted that the aqueous is formed by the ciliary body, but whether it is true secretion as Treacher Collins has written or a filtrate as Parsons has held or whether it is a dialysate as Duke-Elder has so ably expressed his belief still remains an open question, even though Duke-Elder has shown that the relative percentage of the constituents of the ciliary blood plasma and aqueous conforms to the Donnan law of equilibrium. There are certain phenomena that cannot be explained simply on a chemicophysical basis.

*Insufficient Nutrient Material in the Aqueous.*—Weeks has written that it is probable that under certain conditions the supply of nutrient material contained in the aqueous humor is inadequate to sustain the normal metabolic processes in the crystalline lens, or that the food supply, although sufficient, contains substances detrimental to the metabolism of the lens. There may be some obstruction to the proper flow of blood through the minute vessels in the anterior segment of the

globe. Franklin, in discussing Weeks' paper, said that the lens is subject naturally to low grade changes in body metabolism and to systemic toxic influences.

Burdon-Cooper has said that important as diffusion and osmosis are in the nutrition of every cell, they are insufficient alone to account for nutrition of the cell, for it is highly probable that all cells, including those of the lens, appropriate the nutriment by specific affinity, each cell selecting the substance it needs, and only those that have a specific affinity for it can enter. Disturbance of nutrition is the initial factor and cause of cataract. When the nutrition is interfered with, deleterious substances obtained access to its protoplasm, resulting in secondary changes the products of which constitute the opacity.

*Vitamins.*—The relationship of cataract to vitamin deficiency may here be discussed, important as the vitamins are to general and particular nutrition. Pillat, in his observations of patients afflicted with keratomalacia due to vitamin A deficiency, found none with cataract. Recently Day, Langston and O'Brien have reported a complex of cataract and other ocular changes in young albino rats receiving a diet deficient in vitamin G, which is a part of what was previously known as B. No relationship of vitamin C to cataract is known. Vitamin D is known to have a marked effect on calcium metabolism and as such is interesting from the connection of deficiency of calcium to tetany, which is often followed by typical opacities of the lens.

*Abnormal Intra-Ocular Fluids.*—The association of vitreous opacities with cataract leads to the assumption that the fluid surrounding the lens is abnormal. However, cases of cataract do not necessarily show abnormal vitreous. The lens has no blood supply and no mesoblastic tissue; therefore, there is no such thing as phakitis. Recurring uveitis gives rise to cataract.

In persons with heterochromic blue and brown eyes, cataract may develop in the blue eye. If both are blue, the disease is in the eye of the lighter color. Fuchs expressed the belief that there is something connected with the lack of pigment, on the assumption that a disturbance of nutrition is at the bottom of both morbid conditions. It is interesting that in the heterochromic lighter colored eye there is usually evidence of a previous cyclitis. The mere lack of protection from light because of the lack of pigment is not a sufficient cause for the development of the opacity.

*Effect of Light on the Ciliary Body.*—Van der Hoeve expressed the belief that the lens acts to diffuse light, scattering the rays laterally and producing an injurious effect on the ciliary body which causes changes in the quality or quantity of the lens, producing cataract. Changes in the microscopic details of the cells of the ciliary epithelium have been

described in patients with cataract, but they are no different from those found in noncataractous elderly persons, as Duke-Elder has pointed out, nor can the hydropic or degenerative changes that occur in the ciliary epithelium in the diabetic patient be ascribed as the cause of the cataract, as the hydropic change in the lens and that in the ciliary epithelium are both part of the same morbid process.

### III. CHANGES IN THE GENERAL SYSTEM ASSOCIATED WITH CATARACT FORMATION

The idea has been prevalent from the earliest times, as therapeutics shows, that changes in the general condition of the patient have been important in the genesis of primary cataract. There is no question of the advisability of investigating the general condition of the patient, so that any discoverable defect of nutrition or metabolism may be corrected.

*Sclerosis of the Vessels.*—Von Michel saw the cause of cataract in sclerosis of the carotids, which is sometimes unilateral and sometimes bilateral. The want of uniformity of development of cataract in the two eyes he attributed to this variability. The metabolism of the lens is affected by the deficient blood supply. Other conditions associated with disturbances of circulation, such as prolonged ill health and disease conditions of the blood, might act to change the quality or quantity of the nutrient elements in the aqueous. Becker also admitted a morbid condition of the blood vessels. Burdon-Cooper expressed the opinion, however, that there is no need to postulate the existence of a general or local arteriosclerosis in any form whatever, as almost without exception cataract does not follow general vascular hypertension.

*Deficient Function of the Kidneys.*—Fraenkel tested the renal function and found that senile cataract was seldom accompanied by hypertension except when complicated by diabetes or nephritis. Spontaneous cataract does not habitually accompany arteriosclerosis, hypertension or diminished renal function. Fraenkel found changes in the mineral elements of the urine with a considerable diminution of urea and an increase of chlorides; he concluded that the kidney does not eliminate in the ordinary way toxic products elaborated by the organism, but that this inefficiency is unaccompanied by the usual clinical signs of renal deficiency.

Burdon-Cooper also argued for disturbance of the kidneys in cataract because: (1) in evident cases of renal deficiency chemical examination of the lens showed more tyrosine than in senile cataract without renal deficiency; (2) the similarity in the variations from normal between the molecular concentration and surface tension of the aqueous and

similar constants of the urine is so marked that it makes one think that the secretory organ of the eye may have an analogous function to that of the kidney.

*Intestinal Auto-Intoxication.*—Lewis thought that every patient with cataract suffered from intestinal toxemia. Burdon-Cooper found that 80 per cent of them had a well marked indicanuria.

*Diabetes and Disturbances of Carbohydrate Metabolism.*—Berndt, as early as 1834, first reported diabetes as a cause of cataract. Weir Mitchell stated, in 1860, that it is possible that the long-continued presence of even a small amount of sugar may cause changes in the lens productive of opacity. Others have shown since then that the cause was not so much the presence of sugar in the intra-ocular fluids as the vicious influence of the diabetic state on the whole uveal tract.

Duke-Elder differentiated two types occurring in diabetes: (1) senile cataract appearing more frequently and at a relatively early age; (2) more rarely a characteristic form peculiar to the disease.

Weeks said that in his experience of forty-five years he had seen two, perhaps three, such cases. Duke-Elder further stated that probably the main factor in diabetes which determines the high incidence and early occurrence of cataract is the fact that both sugar and acetone sensitize proteins to the denaturing action of light. In addition, the diabetic state, by subjecting the lens to an abnormal fluid traffic, induces the condition still more readily, accelerating the rate of development and tending to alter the type.

Duke-Elder has already suggested that in the rising concentration of sugar in diabetes, provided the available water reserve is maintained, the osmotic pressure of the aqueous tends to decrease; fluid tends to flow into the lens. If the process is relatively mild, the lens swells and is deformed and its curvature and refractive power is increased, and the eye tends to become myopic; if, however, the change is greater and the inflow of fluid more rapid, actual droplets are formed under the capsule, and the transparency of the lens is impaired by the irregular reflection of light at the interfaces between the two media of different refractive indexes. Vacuoles have been noted in the lens in acute diabetic myopia (Lundsgaard and Strickler), and they are seen in typical diabetic cataract (Schnyder). Complete opacity may develop with great rapidity.

*True Diabetic Cataract Is Most Likely Osmotic in Origin.*—It occurs when the concentration of sugar is high, and usually when the patient is fairly well, i. e., when he has a sufficient fluid reserve. It is nearly always bilateral starting immediately beneath the capsule. It occurs frequently in the young: 11 months, Major and Curran (1925); 5 years, Frey (1887); 7 years, Schnyder (1923). It comes on rapidly and

matures in a few days: four days, Scheffels (1898); eleven days, Neubergers (1893). It seldom requires more than a few weeks. With the slit-lamp the symmetrical opacity in the subcapsular region, in the early stage, is seen to be made up of minute, subcapsular, discrete drops of fluid, which are larger at the anterior pole and often confluent at the posterior pole, while the more superficial lens sutures are separated by larger spindle-shaped lacunae. Fischer reported a case of diabetic cataract in a girl of 17 years. The symptoms disappeared after prolonged treatment with insulin. A few other similar experiences have been recorded.

*Dextrose Metabolism in Patients with Senile Cataract.*—Baldwin and Bartel, in 1924, reported a study of 132 consecutive cases of cataract. They found that 99 of the 132 patients had a blood sugar of over 120 mg. per hundred cubic centimeters. H. Maxwell Langdon, in 1925, published a report on chemical studies of the blood in patients with senile cataract. In the 100 cases included in his series, 15 showed a high blood sugar and 45 a lowered dextrose tolerance.

In 1928, O'Brien published a report on studies of dextrose metabolism in patients with senile cataract. In this series of seventy-four consecutive cases, the fasting blood sugar level was above 120 mg. per hundred cubic centimeters in 82 per cent, and there was a lowered tolerance for dextrose in 45 per cent of cases.

O'Brien, in a further study (1931), stated that a hyperglycemia existed in approximately one half of the cases of a second series of 218 patients with advanced senile cataract. He expressed the belief that abnormally high concentrations of sugar in the blood and body fluids may have a bearing in many cases on the etiology of cataract.

Woods' basic criticism of O'Brien's hypothesis was the definite lack of controls. He thought that the way to approach a proposition such as O'Brien brought forward was to have two parallel series, one of nondiabetic, noncataractous persons and the other of nondiabetic, cataractous persons of approximately the same ages throughout. Parallel estimations of both the blood sugar and the fasting blood sugar taken at different times of the day over a prolonged period of time would give two comparable sets of figures which should easily settle the question of whether or not the cataractous group handled their sugars in a different manner than the noncataractous group.

I believe that the finding of hyperglycemia in elderly persons with cataract is simply another expression of the diminished functions of old age, but still its correction offers a mode of approach for the treatment of patients and is bound to be beneficial as far as the local nutrition and maintenance of transparency of the lens are concerned.

The cataract of cholera is similar to that of diabetes. It is most probably due to abstraction of water from the lens (general dehydration, Fuchs), but Duke-Elder expressed the belief that the opposite process may be at work. The mucosa of the bowel is disorganized and allows the passage of material from the blood, in the order first of water and salts and later organic substances and proteins. This early loss of the dialyzable and osmotically active constituents, while rendering the blood more viscous, will make it more dilute in the osmotic sense, and if the fluid volume is at all kept up, water will tend to flow into the lens.

*Disturbances of Fat Metabolism.*—The lens is entirely of epithelial origin. Owing to protein disintegration, there may be formed or deposited secondarily certain fats or products of fat metabolism. Cholesterol, from 6 to 7 per cent, has been found in cataracts by Zehender and Mattieson; also by Jacobson, Kuhne and Leber. Burdon-Cooper found more cholesterol in diabetic cataract than in any other type. It represents most probably a reduction product of a previously formed acid. O'Brien (1928) found blood lipoids increased in 52 per cent of cases of senile cataract (normal, from 0.6 to 0.7 per cent). Cholesterol was increased in 50 per cent (normal, from 0.14 to 0.17 per cent). Lecithin was increased in 70 per cent of cases (normal, from 0.2 to 0.3 per cent).

Heath stated, in general, that lipoids (except soap) are insoluble in water. Their presence in the living cell is necessary. They are normally found in substantial percentages in blood and tissue in a state of fine emulsion and balance. Certain metabolic reactions, such as the chemistry of aging, hereditofamilial and endocrinologic factors, vascular lesions and stasis, disturb this emulsion. Its proportions and concentration are altered. The emulsion coarsens, and the larger particles become foreign bodies in effect.

Friedenwald has said that there is always a tendency to incriminate an altered fat metabolism, but that at the present time there is no evidence to warrant it as the cause of cataract.

*The Influence of Tetany on the Aqueous and the Development of Cataract.*—Weiss was the first investigator who reported the development of cataract after the extirpation of goiter. In his first publication he mentioned three cases, and in 1883, thirteen cases.

Sandstroem in 1880 discovered the parathyroid glands. Vassale and Generali came to the conclusion that extirpation of the parathyroid glands caused tetany, while extirpation of the thyroid gland alone did not bring about tetany, but cachexia.

Unfortunately this theory was ignored by surgeons for a long time, until Wedheim, by surgical experiment, proved it and found that cataract developed in tetany.

*Cataract Associated with Convulsions.*—Formerly, according to von Arlt (quoted by Fuchs), cataract was considered to be caused by convulsions, because many of the patients had suffered from them in early youth. The opacity is produced by minute spherules of material deposited after vacuole formation either as a process of faulty nutrition or metabolism or through faulty fiber formation or fiber degeneration. These changes occur in other epithelial structures, such as the teeth, nails and hair.

*Tetany Cataract.*—Peters (quoted by Siegrist) in 1898 demonstrated two women of middle age, one of whom suffered from cataracta nuclearis, the other from right total cataract. Both had suffered from typical tetanic convulsions. There is only the possibility, said Peters, that cramp of the ciliary muscles causes a disturbance in nutrition. The food supply becomes inadequate, and the nucleus shrinks. Von Hippel replied that if such is the case, the action of strong doses of physostigmine (esterine) should also cause opacity of the lens. To this Peters answered that toxic quantities of physostigmine would be necessary, but according to Siegrist this reply is not satisfactory. Thirty-eight cases of known postthyroidectomy tetany with cataract have since been published.

Eiseman and Luckhardt simulated the symptoms of parathyroid deficiency in dogs, producing tetany by intravenous injections of oil of wormwood and by subcutaneous injections of strychnine sulphate, without producing cataract in any of their animals. They also lowered the blood calcium by intravenous injections of sodium oxalate and sodium acid phosphate without producing cataract. In nearly all the clinical histories of the patients with postthyroidectomy cataract, however, there have been convulsions before the opacities of the lens developed. There have also been reported occurrences of ergot poisoning followed by cataract. The action of ergot is on smooth muscle, affecting probably the smooth muscle of the ciliary body to produce the interference with the formation of aqueous.

*The Mechanism of Tetany Cataract.*—It is difficult to prove the mode of action of the decreased calcium content of serum and necessarily of the aqueous, vitreous and lens in the pathogenesis of tetany cataract. While this total amount is extremely small, the electrochemical changes and the disturbance of equilibrium, depending on the loss of the calcium ions, are out of all proportion to the chemicals involved. The decreased calcium content can certainly cause changes in the labile protein of the lens. Processes of hydrolysis or hydration may produce the globules, vacuoles and cloudy swelling of the lens fibers.

*Calcium Metabolism of Persons with Senile Cataract.*—Fischer and Triebenstein examined sixty-eight cases of senile and presenile cataract

and claimed to have found evidences of tetany and latent tetany in 88.2 per cent. E. Tron and Pellathy and Polichova, using different methods, studied the blood serum calcium in patients with senile cataract, but could not confirm the association with parathyroid insufficiency. There was, however, considerable difference in their results, but they indicated an excess rather than a deficiency in cases of cataract.

*Calcium Content of Lens Increased in Mature Senile Cataract.*—Burge found that the calcium in the crystalline lens increased from a very small amount in the normal lens to a relatively large amount in cataract, 12.5 per cent of the ash of the cataract. Adams found the calcium content of the normal lens 0.0516 Gm. per cent of the dry weight, while in cataract it was 0.1256 Gm. per cent of the dry weight.

The presence of increased calcium in cataract is either (1) primary and an actual etiologic factor in producing opacity of the lens or (2) secondary to local tissue changes, which exist in the lens regardless of the calcium content of the serum, aqueous or lens. If it is primary, it is interesting to speculate on the possible mode of action in the production of the opacity. It might act: (*a*) In reducing the permeability of the capsule. Friedenwald demonstrated that in vitro calcium has this action, and he has shown that under other conditions it takes only a relatively slight reduction in the permeability of the capsule to obstruct the metabolic processes of the lens sufficiently to cause cataract. (*b*) In sensitizing the protein of the lens for an abnormal action of ultraviolet rays. Burge found that in vitro the protein of the lens could be so influenced by calcium that ultraviolet rays would act to precipitate it, but he concluded that the concentration of calcium and the amount of light necessary to produce this in vitro are not found in vivo. (*c*) In altering the colloidal system of the lens, so that precipitation of the protein occurs. Here again Adams found that the conditions necessary for this reaction are not met with in the living body. Finally, it must be remembered that there is no direct evidence that the calcium of the lens is increased before the cataract forms.

*Deposition of Calcium a Secondary Reaction.*—Salit and Mattill reported that the examination of five human lenses in the early stages of cataract did not show any calcium. This finding is important in ruling out an excess of calcium as a factor in initiating the cataractous process.

One must feel for various reasons that the increase of calcium in cataract is not primary, and that it is not the cause of cataract, but that it is a coincident or secondary change that takes place with or follows other alterations in the lens. The calcium is adsorbed to the large molecules of the lipoids, and the calcium is then deposited by a change in the $p_H$ toward the alkaline side.

Evans and Kern, in their experiments, produced cataracts by removing the parathyroids, and found changes in the mineral content of the cataracts, in calcium, magnesium, potassium, phosphorus and silicon, as compared with the normal, similar to the changes described by Burge in human senile cataractous lenses. Therefore, they expressed the belief that serious consideration should be given the matter of dysfunction of the parathyroid glands in the etiology of senile cataract. I found, however, that there is no evidence of calcium deficiency as far as the values for blood serum are concerned in cases of senile cataract.

*Endogenous Calcium Metabolism of Patients with Senile Cataract.*—In a study of the endogenous calcium metabolism in a small series of cases of senile cataract, the relation of the intake of calcium in the diet was carefully calculated and compared with the output of calcium in the urine and feces. The results showed that the patients were in perfect or slightly positive balance when on a neutral or slightly alkaline diet, but when on an acid residue diet or receiving injections of parathyroid extract, they were in negative balance, excreting more calcium than they ingested. In other words, the patients were responding normally to the factors influencing calcium metabolism and were utilizing their calcium properly.

*Dysfunction of Other Endocrines.*—Siegrist has advanced his hypothesis based on endocrine dysfunction. The importance of endocrine secretions in cataract has been proved by the removal of the parathyroid glands. The whole body is influenced by endocrine secretion, and youth and age itself are merely stages of its function. It is improbable that the lens is an exception. Steinach has definitely proved, by his operation, that the gonads influence youth and age. Harms said that he repeatedly saw that after the transplantation of a testis in dogs cataract disappeared and the lens became clear. However, Siegrist expressed the belief that this has no value, because ocular examinations done by investigators other than oculists are not reliable.

Asher (quoted by Siegrist) found that up to 50 years of age men and women suffer alike from cataract. After this age, women are more often afflicted with cataract. He examined the eyes of twenty-four women whose ovaries had been removed, and found that all of the lenses were clear, with the exception of four in which there were small, equatorial opacities. Asher further said that there is no reason to presume that cataract should be due to the cessation of ovarian function. Siegrist said that the influence of slow, senile degeneration of the ovaries on the organism need not be identical with the sudden cessation of all function by the total removal of the ovaries. Gallus (quoted by Siegrist) expressed the opinion that the climacterium in women favors the formation of cataract.

*Myotonia Atrophica.*—Fischer (quoted by Siegrist) closely watched thirty-eight patients with myotonia atrophica, 92 per cent of whom suffered from cataract. Vogt was very much interested in this condition. He found that in eight lenses, removed from five persons, the opacity was caused by masses of small spots in the cortex which he thought might perhaps have been cholesterol. J. Hoffmann (quoted by Siegrist) described five cases, and referred to the ancestry of one family with myotonia atrophica associated with cataract. However, his own conclusions were that there is no special connection.

Curschmann (quoted by Siegrist) said that it is easily believed that auto-intoxication is the cause of cataract, especially in myotonia, and that one should watch endocrine secretion more closely. There also may be a relationship between the lens and the nervous system. In one of his cases myotonia started with symptoms of tetany.

Weeks stated that the absence of the necessary elements in the fluids of the body for the proper nutrition of the affected atrophic tissues evidently is also responsible for the changes in the lens. The same idea most probably holds for the production of cataract in cases of scleroderma.

### RÖMER'S THEORY OF SPECIFIC CYTOTOXINS

The conjectures of Römer as to the action of specific lentotoxins in causing cataract are interesting. He compared the lens to a red corpuscle having a surrounding membrane which impedes the diffusion outward of important constituents. When the hemoglobin has passed out of the erythrocyte, the cell is dead; so, too, when in cataract the soluble proteins have disappeared from the lens, it is an expression of the death of the lens fibers. Previous observations in hemolysis have shown that isohemolysins can be formed, e. g., in the goat (Ehrlich and Morgenroth); auto-hemolysins, on the other hand, are formed only under pathologic circumstances. If autocytotoxins occur, it might be expected that they would be a sign of senile change, for atrophy and degeneration of tissues are the common features of old age. If lentotoxins occur, it is necessary that they are not able to reach the lens, or rather that the lens is protected from them under normal circumstances. It is only when the ciliary epithelium is deranged that they go through into the aqueous. There they may be able to enter the lens and act by specific fixation of the lens cells or protoplasm. As soon as this occurs, the cells are poisoned or the protoplasm changes so that abnormal osmotic conditions prevail and opacity is produced.

Mijashita and Bingers (quoted by Siegrist) showed that there is no absolute retention of cytotoxin, because receptors of the third order pass into the humor of immunized animals. Siegrist said that Römer did not succeed in proving or in substantiating his theory. The lens

contains more substances, but it is not known yet what they are, how they act or interact and what significance they have.

Duke-Elder stated that Römer was wrong about the appearance of immune bodies in the normal aqueous, as they have been demonstrated only in highly immunized animals.

Burdon-Cooper expressed the belief that the presence and accumulation of cytotoxins in the organism are probably a constant physiologic phenomenon without detriment to the lens, unless there is inefficient destruction or elimination of the cytotoxins.

*Naphthalene.*—Salffner found in naphthalene cataract an admission of water as the first sign of change, owing most probably to an inhibition of the aqueous. Burdon-Cooper expressed the opinion that naphthalene cataract demonstrates the fact that specific cell poisons of the lens exist. He said that chemically defined protoplasmic poisons do exist, but that he did not know their relation to senile cataract. It is possible, under the conditions of metamorphosis of the body, that such bodies and antibodies appear. Once senile involution has started, there must be regulative devices to prevent the formation of noxious products of metabolism or to counteract them. If these fail to a greater or lesser degree, then there appear certain products that may injure the lens.

*Complicated Cataract.*—Duke-Elder is of the opinion that complicated cataract is probably caused by malnutrition and the action of toxins, acting directly on the lens substance or indirectly by altering the permeability of the capsule. It occurs typically in slow chronic inflammatory conditions in the posterior cortex, because here the capsule is thinnest, permitting diffusion more readily, and the vitreous membrane is attached here.

Healy (1921) felt that the central hyaloid lymph canal may exert a decidedly localizing influence in carrying the toxins to the posterior cortex, where complicated cataract usually begins.

## SUMMARY AND CONCLUSIONS

Representative and significant hypotheses concerning the pathogenesis of cataract have been reviewed and quoted. These have been arranged in three groups: (1) that in which the condition is dependent on changes in the lens itself; (2) that in which a physicochemical theory is advanced, that the nutrition of the lens is disturbed by changes in the quality or quantity of the nutrient material supplied to it by the ciliary body, and (3) that in which the cause is said to be dependent on some general disease or on a metabolic or nutritional disturbance.

This survey has been made in order to record all of the worthwhile thoughts on this complex problem. The very number of ideas presented proves that there are many phases that are not properly explained

by any one hypothesis. Each one adds something, however, to the knowledge, and probably with further advances and correlation much that is now obscure will, in the future, be elucidated.

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# Correspondence

## "HOMATROPINE AND ATROPINE CYCLOPLEGIA"

*To the Editor.*—May I express my thanks to Dr. Linn Emerson (ARCH. OPHTH. **7:** 954 [June] 1932) for calling my attention to his article in the *Medical Digest* of October, 1904, and for the copy of the tabulated results? I am indeed sorry that I did not find any reference to it, though I went back as far as 1880 in my search through the literature.

I am sorry that I could not tabulate the individual findings in the 1,260 eyes, as they would reveal the comparison much more vividly. It would have made my article (ARCH. OPHTH **7:** 294 [Feb.] 1932) much longer than the editors would care to publish.

First, in reply to Dr. Emerson's criticism of the homatropine procedure, allow me to say: We have been using fresh solutions (never over ten days old) of Merck's homatropine hydrobromide (2 per cent). I have tried it in combination with 1 per cent cocaine and found that cocaine did not add to the cycloplegic effect. I agree that the so-called drying effect of cocaine is negligible, for, I, too, have found that the patients can often read 20/15 while under the effects of homatropine and cocaine. In my experience, there has been no more conjunctival irritation, spasm or photophobia without cocaine than with it added to the homatropine, and for these reasons, I use homatropine alone.

Using homatropine six times, we have found, gives maximum cycloplegia, if the retinoscopy is done ten minutes after the last drop. This time was arrived at by Dr. E. V. L. Brown many years ago and more recently by myself, when we found that repeating the retinoscopy at longer intervals gave varying results. It has been found in Dr. Brown's clinic, at the University of Illinois and at the University of Chicago, that in training beginners in retinoscopy, the findings are at such variance under homatropine that even well trained observers cannot check each other within 0.50 diopter; while in cases in which atropine is used, checking can be so accurate that beginners are required, before they are passed in retinoscopy, to come within 0.25 diopter in ninety of a hundred consecutive cases, and most students do even better than this. In our clinic, using homatropine, trained refractionists are able to check within 0.25 diopter within ten to twenty minute after the last drop is instilled, but not if a longer period elapses, unless homatropine is continued at ten minute intervals. The fourteen drops of atropine were adopted by Dr. Brown after experimenting to learn the minimum of atropine that gives the maximum cycloplegic effect. More than that amount does not give a greater cycloplegic effect except in cases of real ciliary spasm.

I am at a loss to understand such close approximation of Dr. Emerson's figures for children. Would not the variance have been far greater in a larger series of cases? In my study, I found the greatest differences in children.

I cannot agree with the statement that scopolamine is a better cycloplegic than atropine. I have used scopolamine and found it better

than homatropine, but with less cycloplegic effect than atropine. Then, too, the toxic reaction seen all too frequently, even though pressure is made over the tear sacs, occurs too often for universal use. I have seen patients mumble incoherently and stagger out of the clinic or office after its use too often to want to employ it as a routine procedure.

If the difference between the results with homatropine and atropine was only 0.25 or 0.50 diopter, I could find no excuse for the publication of my results or for championing the use of atropine in refraction. We have found, however, that all too frequently patients with unaided vision of 15/10 when under homatropine will show on retinoscopy the need of a weak minus lens or a plus 0.25 sphere or cylinder—in other words, a glass which is entirely too weak to explain the symptoms—but when refracted under atropine need a plus 1.00 or more sphere with or without cylinders and, by using such a glass for reading, get the desired relief. It seems that this is more often found in patients with dark brown irides. We all know what questionable findings we get with homatropine in colored patients.

Scarcely a week goes by that we do not see a patient wearing weak minus lenses who has had homatropine refractions and is having trouble with near vision. Such patients usually show the need of a hyperopic or mixed astigmatism correction under atropine and are free from symptoms when wearing the atropine correction for reading.

A study of table 1 in my article showing the *change* in the *type* of refractive errors in ninety-eight eyes, I think, should be conclusive proof that atropine is a better cycloplegic. I am now studying an additional hundred cases of low myopia and emmetropia under homatropine which show a change in the type of refraction under atropine.

In conclusion, I shall say that we do not use atropine as a routine measure in all cases of refraction. We do a few manifests and a large number of homatropine refractions. We use atropine in all children under 16 years, in practically all persons with myopia, in all young persons with hyperopia with better than 12/10 vision, in those complaining of headaches and in all cases in which we do not get satisfactory relaxation under homatropine regardless of age.

LOUIS BOTHMAN, M.D., Chicago.

## "THE IOSKIASCOPY TEST"

*To the Editor.*—In my article on "The Ioskiascopy Test" (ARCH. OPHTH. 7: 378 [March] 1932) two numerical errors crept in, and I would request you to publish the corrections. On page 381, line 27, where the new position of the arrow-tip is given as "no. 25 on the inner scale," this should read "115 degrees on the inner scale." It is 25 degrees on the outer scale, but this scale is not used with minus cylinders. On page 382, line 13, where the new position of the arrow-tip is given as "no. 135 on the outer scale," this should read "45 degrees on the outer scale." It is 135 degrees on the inner scale, but this scale is not used with plus cylinders.

I may add that in locating the new position of the arrow-tip from the tables according to the system described in the article, the horizontal lines represent the true axis and the vertical lines represent the false axis.

JOSEPH I. PASCAL, M.D., Boston.

# Notes and News

EDITED BY DR. JOHN HERBERT WAITE

## GENERAL NEWS

**New York Academy of Medicine: Graduate Fortnight.**—The New York Academy of Medicine invites the medical profession of the country to participate in the intensive two weeks' study of tumors, benign and malignant, to be held from October 17 to 28, inclusive. A full program of clinical demonstrations, lectures and conferences has been arranged to cover all phases of the subject, diagnosis and treatment. There is no charge for attendance at any of the clinics or meetings, but registration is required for participation in the hospital demonstration clinics. A complete program and registration blank for the 1932 Fortnight may be secured by addressing the New York Academy of Medicine, 2 East One Hundred and Third Street, New York.

**New Italian Ophthalmologic Journal.**—The first number of a new Italian journal devoted to ophthalmology has just come to hand. It is Rassegna Italiana d'ottalmologia, founded and edited by L. Guglianetti, Professor of Ophthalmology at the University of Turin. He is assisted by Professors Angelucci and Cavara of Naples, Lo Cascio of Padua, Ovio of Rome, Pes of Genoa, de Lieto Vollaro of Milan and others. The journal was founded, according to its editor, because of the large amount of research in ophthalmology being conducted in Italy. It will appear in monthly numbers of about a hundred pages or bimonthly double numbers, and will include original studies, clinical and therapeutic notes, general reviews, abstracts, society reports and announcements. The abstracts will cover all Italian ophthalmologic articles and the most important foreign ones.

Original articles in the present double number for January and February include a histopathologic study of argyrosis affecting the tarsus by C. Gallenga, researches on trachoma by Favaloro, a report of a case of congenital lacrimal fistula by Tirelli, a study of the disagreement of the two eyes in reading by Ovio, the prismatic effect of decentration by Vita and an extensive embryologic and anatomic study of the so-called lacrimal muscle of Duverney-Horner by R. Gallenga. These articles will be reviewed later, but the character of them shows the type of work being conducted in the Italian clinics. The fact that this will be the fourth Italian journal devoted to the publication of such work indicates the activity of these clinics.

Not only in such great centers as Rome, Naples and Milan, but also in at least fifteen smaller cities, such as Padua, Palermo, Catania and Pisa, universities with well organized ophthalmologic clinics exist. The university system resembles that of Germany more than that of other Latin countries, with professors, instructors and assistants who receive promotion from one clinic to the other in reward

for their scientific work. Thus a constant stimulus to investigation is afforded, which results in a great amount of research. While the quality of such work is of necessity uneven, a number of subjects have received most thorough and satisfactory study in these clinics, representing definite advances in ophthalmologic thought.

The present journal is well printed on good paper, with numerous excellent illustrations in black and white and in colors. It will no doubt be of a similar character to the excellent Italian journals already existing and the ARCHIVES OF OPHTHALMOLOGY takes pleasure in offering congratulations on its auspicious beginning.

**Summer Graduate Course in Ophthalmology.**—Beginning Monday, Aug. 1, 1932, the Department of Ophthalmology of the University of Rochester will conduct the third annual summer graduate course of lectures and demonstrations in ophthalmology. The course will cover a period of five days, and will be limited to fifty members. The subjects to be covered are: ophthalmic surgery, ophthalmoscopy and medical ophthalmology, perimetry and ophthalmic neurology, slit-lamp microscopy, ophthalmic myology, pathology, retinoscopy and refraction. The visiting lecturers will include: Dr. E. V. L. Brown, Chicago; Dr. Henry P. Wagener, Rochester, Minn.; Dr. C. S. O'Brien, Iowa City; Dr. Dorland Smith, Bridgeport, Conn.; Dr. Algernon B. Reese, New York; Dr. John N. Evans, Brooklyn, and Dr. Francis H. Adler, Philadelphia. Inquiries may be addressed to Dr. John F. Gipner, Strong Memorial Hospital, Rochester, N. Y.

**Ophthalmic Examinations.**—The American Board for Ophthalmic Examinations will hold an examination in Montreal on Monday, Sept. 19, 1932, at the time of the meeting of the American Academy of Ophthalmology and Oto-Laryngology. Necessary applications for this examination can be procured from the secretary, Dr. William H. Wilder, 122 South Michigan Avenue, Chicago, and should be sent to him at least sixty days before the date of the examination.

## SOCIETY NEWS

**Central Wisconsin Society.**—On June 4, the Central Wisconsin Society of Ophthalmology and Oto-Laryngology was addressed by Dr. Louis Bothman on "Cataract Surgery in India" and on "Detachment of the Retina, Diagnosis and Surgical Treatment." Dr. Francis Lederer spoke on "Mastoiditis, Its Pathology and Indications for Operation," and "The Present Status of Malignancies About the Head and Neck."

**International Ophthalmological Congress.**—It is desired to correct directions previously given with regard to abstracts and discussions of papers. These communications should be addressed to the Secretary of the National Committee at Madrid, Dr. Francisco Poyales, Olozaga 3, Madrid, and they should reach him before Nov. 1, 1932.

# Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

## Anatomy and Embryology

THE DETERMINATION OF LINEAR MEASURES ON THE SURFACE OF THE GLOBE. H. WAGNER, Arch. f. Ophth. **127:** 103, 1931.

The location of the ora serrata can be determined on the living human eye by transillumination with Lange's lamp. The method reveals a dark ring a few millimeters behind the dark portion of the ciliary body. This ring is crescent-shaped on the temporal side, and its posterior edge indicates the position of the ora serrata. On the nasal side only a narrow somewhat wavy line is visible in that region. Eyes with a moderate amount of pigment (light brown eyes) show the ring better than very light or very dark eyes. Histologically, the ora serrata is characterized by a thickening of the pigment epithelium and by the formation of pigment processes sticking out into the underlying tissue.

The linear measures on the surface of the globe were obtained on eighteen eyes enucleated shortly after death. The distance between the limbus and the ora serrata was found to vary with the axial length of the globe. For an axial length from 24.7 to 26 mm. it was found that the ora serrata-limbus distance was from 6 to 7 mm. on the nasal, and from 8 to 9 mm. on the other side. In eyes with an axial length between 22.7 and 23 mm., the same distance was from 4.8 to 6.5 on the nasal and from 6 to 7.5 mm. on the other side. In eyes with an axial length of from 23.5 to 24.5 mm., there was a range of from 6 to 8.5 mm. for the ora serrata-limbus distance.

The author also determined the distance from the posterior pole and from the papilla to the limbus in eighteen eyes. For the surgeon who attempts the localization of retinal tears it is of great value to be able to project the ora serrata accurately on the surface of a living eye by means of transillumination or diaphanoscopy. P. C. KRONFELD.

PLASTIC ANATOMY OF INFANTS' AND CHILDREN'S EYES. R. H. KAHN, Med. Klin. **28:** 365 (March 11) 1932.

The report of W. Reitsch concerning the artist's unusual conception of the eyes of children and infants stimulated the author to take photographs of many children's eyes from the time of birth at intervals to the age of 15 years. Reitsch felt that no infants' eyes were depicted in the most famous canvases. The author takes exception to the statement, as he believes that the children in Andrea della Robbia's "Foundling Home" are from 2 to 3 years of age. He also cites the fact that in Baroccio's "Prince of Urbino," the eyes are those of a 5 or 6 year old child. In "The Holy Family" of Roger van der Weyden, in which the Christ child is depicted as an infant, the eyes are those of a much older child. On the contrary, the infant in the picture, "The Birth of Christ," by Micheal Packer, shows deep in the fissure the erected infant's eyes.

A series of photographs accompanies the article showing the eyes of a single person taken at the following ages: 48 hours, 11 weeks, 21 months, and 2, 3, 8 and 15 years. The changes in the lids, globe and fissures are explained in detail for each age. L. L. MAYER.

## Bacteriology and Serology

IMMUNE-BIOLOGIC REACTIONS ON THE QUESTION OF THE HYPERSENSITIVITY TO LENS PROTEIN IN MAN AND ANIMALS. R. BRAUN, Arch. f. Augenh. **105:** 122, 1931.

The author first reviews the literature on the experimental production of precipitins to lens protein and discusses the question of organ specificity of the lens and the clinical question of endophthalmitis-phacoanaphylactica. He then reports his own experimental work, which may be summarized as follows:

1. The injection of foreign lens protein into rabbits failed to produce precipitins, and the author therefore believed that lens protein was an excessively poor antigen.

2. He found, however, that by sensitization and later intoxication with foreign lens protein definite anaphylactic reactions could be produced in guinea-pigs.

He concludes: (1) that the production of precipitins is not possible either with foreign or with homologous lenses in rabbits and guinea-pigs, and (2) that the injection of foreign lenses will produce a hypersensitivity, and that allergic reactions can be evoked by a later shocking injection of such foreign lens protein. This, however, is not the case with homologous lens protein.

In the second portion of his paper he investigates the question of hypersensitivity to lens protein in man, and here uses the same general technic of intradermal injection which has been outlined by various American authors. The lens protein used in this case was swine lens protein, and as a control the author used horse serum. He found that 75 per cent of persons negative to the intracutaneous test with horse serum were likewise negative to the intracutaneous test with swine lens. On the other hand, 86.7 per cent of persons sensitive to horse serum were also sensitive to swine lens. He therefore concludes that the intracutaneous test with pig lens is too greatly influenced by nonspecific factors to permit its being of any clinical value.

The third portion of his investigations are concerned with the possibility of auto-iso antibodies to lens protein in the serum of cataractous patients. Unable to demonstrate precipitins in the serums of such patients, he resorted to a passive sensitization experiment. In this experiment guinea-pigs were given intracardial injections of the patient's inactivated serum, and six minutes thereafter 0.5 cc. of the patient's lens by the same intracardial route. In the majority of such guinea-pigs, the author noticed a definite fall of temperature, which he thought suggested anaphylaxis due to the passive sensitization. He feels, however, that these experiments were inconclusive, but that they offered the only entity on which one could base the theory of endophthalmitis-phacoanaphylactica.

These conclusions are so radically in conflict with those of other workers in this field that they will undoubtedly meet with prompt criticism.

F. H. ADLER.

PNEUMOCOCCIC INFECTIONS OF THE EYE. E. LOBECK, Arch. f. Ophth. **127:** 395, 1931.

The pneumococci obtained from diseased eyes were classified. Strains that were not agglutinated by any of the three known serums were further analyzed by intraperitoneal injection into mice, which permitted the differentiation between a virulent and an avirulent type IV. In cases of simple conjunctivitis, the avirulent type IV was most frequent. Pneumococci of the type II were found in no case; type I in one case of serpiginous ulcer. The remainder of the cases with serpiginous ulcer or chronic dacryocystitis showed either type III or the virulent type IV. This explains why the serum therapy of eye infections with pneumococci failed, because there are therapeutic serums only for type I and II.

P. C. KRONFELD.

## Cornea and Sclera

INTERNAL ULCER OF THE CORNEA. C. MARIOTTI, Arch. di ottal. **38:** 586 (Nov.) 1931.

Previous cases of this condition, a loss of substance of the posterior parts of the cornea, with defects in Descemet's membrane, are reviewed. The name has been used to describe a somewhat variable picture in which two types of condition may be distinguished, the simple loss of substance met with in interstitial keratitis, and the keratitis pustulosa profunda of Fuchs which usually accompanies iritis and, like the first type, is often of syphilitic origin.

The author's patient was a man of 37, whose right eye had undergone several severe attacks of inflammation during sixteen years. A large corneal scar was present, with several old posterior synechiae; vision was reduced to perception of movements of the hand by a membrane in the pupil. Some active infiltration of the remaining clear cornea was present. On account of pain the eye was removed. Sections showed the posterior corneal surface to be covered by a layer of new connective tissue. In the central region Descemet's membrane was absent, and a large defect in the stroma was occupied by a mass of round cell infiltration showing some necrosis in the center and projecting into the anterior chamber. The iris and the ciliary body showed active infiltration.

Syphilis and other general infections could be excluded, and the author believes that an injury to the cornea was the primary cause of the condition, opening the way to organisms from without or to an endogenous tuberculous infection. Examination for organisms, including tubercle bacilli, gave negative results.

S. R. GIFFORD.

FORMATION OF MARGINAL FURROWS AND ECTASIAS OF THE CORNEA. W. THAMM, Klin. Monatsbl. f. Augenh. **87**: 44 (July) 1931.

The formation of a marginal furrow and ectasia of the cornea rarely prompts a patient to consult an oculist, because of the absence of inflammation and visual disturbances. The pathologico-anatomic features of this condition are discussed by the author, who reports the case of a man, aged 52. He was operated on for bilateral pterygium in 1908, but had no other pathologic disturbances, except slight protracted irritation of the conjunctiva, until recently, when the vision in his left eye decreased. His right eye had been weak for many years; its cornea showed a slightly brownish tinge along the upper and nasal portion of the gerontoxon, and a clear furrow which was thinner than the remainder of the cornea. Numerous yellowish deposits were observed in the layers of the cornea; vision was greatly reduced owing to changes in the macula. The left eye presented a very thin and transparent ectasia, besides the same marginal furrow described in the right eye. The vision was normal for distance and close range, after correction of astigmatism of about 4 diopters.

Fatty degeneration at the marginal portion of the cornea, with deposits of fat near the limbus, which block nutrition, is considered the cause of this malformation. It is impossible to decide whether the process leads to simple fatty infiltration of hitherto healthy cells, or if it is a degeneration of diseased cells; inflammation can be excluded as an etiologic factor. The striking transparency of the pit of the furrow is explained as marginal atrophy.

The therapy is powerless, although excision, or cauterization, of the thinnest ectatic portion, combined with covering by a conjunctival flap, may prevent perforation or the formation of excessive astigmatism in some of these cases.

K. L. STOLL.

EXPERIMENTAL AND CLINICAL OBSERVATIONS ON TUBERCULIN REACTIONS IN TUBERCULOUS KERATITIS. A. J. SAMOJLOFF, Klin. Monatsbl. f. Augenh. **87**: 215 (Aug.) 1931.

The clinical aspects of tuberculous keratitis offer no characteristic differences from simple parenchymatous keratitis of syphilitic or other origin. Neither slit-lamp examination nor Pirquet's test, independent of other diagnostic means, can furnish any diagnostic proof. Mantoux' test appears to be more reliable. The author performed a series of experiments on animals with a view to studying the effect of injections of tuberculin on the tuberculous focus in the cornea. Only animals in which the corneal conditions were experimental primary tuberculous foci were used. Samojloff found that the tuberculin therapy caused reactions, not only in the corneal foci, but also in the other tissues of the eye, especially in the uvea. Two animals with experimental tuberculous keratitis received injections of sterile milk for control. They developed no decrease of the intra-ocular tension, nor other symptoms of local or general tuberculous reaction, the course of the corneal condition was in no way influenced, and extravasations could not be discovered with the slit-lamp. The reaction, therefore, differed from that

obtained with tuberculin. The effect of the subcutaneous injections of tuberculin in these animals was so striking that sixteen patients with diffused tuberculous interstitial keratitis received the same treatment. The general character of the focal reaction was similar to that of the animals, but less severe. The temperature rose simultaneously with the first focal reactions in only one third of the cases. Biomicroscopically visible reaction confirmed the tuberculous nature of the corneal disease and furnished an opportunity to recognize the general immunobiologic condition of the body, which is very important in the therapeutic application of tuberculin. Samojloff is convinced that the reaction of the ciliary body, manifested by decreased intra-ocular tension, is a constant and characteristic symptom after injections of tuberculin. It is necessary to watch even a small increase of the temperature which would forbid further injections, unless it were accompanied by an active ocular reaction. The fifteen patients received no local treatment of their eyes; the tuberculin treatment lasted from three to eight months. A cure may be obtained even in cases of keratitis with apparently complete cicatrization, as long as the least biomicroscopically visible activation of the process, i. e., focal reaction in the cornea, can be effected.

K. L. Stoll.

## General

Self-Help in Seeing by a Four Months Old Child. S. Engel, München. med. Wchnschr. **78:** 2149 (Dec. 18) 1931.

The author reports the case of a premature infant with a rare condition, and his reaction to it. At birth the child weighed only 1½ pounds (680.4 Gm.), and had an unusually large head, an overhanging forehead, a rachitic rosary, slight craniotabes and a protruding abdomen. The eyes, including the pupillary reactions, fundi and refraction, were normal. The outstanding feature was the lids. Because of the enlarged head with its horizontal widening, the lower lids were stretched, so that instead of the usual concave form being presented to the globe, there was a straight line. Also, the outer portions of the lower lid were distorted upward. In looking down, the lower lid, instead of being drawn slightly downward, had a tendency to entropion. The condition was bilateral and did not disturb the child when he looked straight ahead or upward. However, when he looked toward the foot of the bed, the entire pupillary areas were covered by the lower lids. The case was not typically rachitic with hydrocephalus, as the globe was normally placed and no sclera was visible above the limbus. At the age of 4 months it was noted that in order to see downward the child pulled the lower lid out of the way with his hand. The left hand was the one of choice in this maneuver, although the right hand could be brought into use by fixing the left hand. This phenomenon is not described in the pediatric handbooks. In the foundling clinic in cases of chronic hydrocephalus such a condition is characterized by saying that "the child puts on the glasses." The author comments on the mechanism of the child's reaction. Two excellent photographs accompany the article.

L. L. Mayer.

## Glaucoma

PRIMARY GLAUCOMA AND THE ORGANIC CONSTITUTION. V. ROSSI, Arch. di ottal. **39:** 1 (Jan.); **39:** 51 (Feb.) 1932.

The author reviews the relationship between glaucoma and various general factors, such as race, sex, age, endocrine balance and the chemical composition of the blood. The factor of heredity is not proved for glaucoma as a disease, but only in the inheritance of various conditions predisposing to glaucoma. As regards the ductless glands, the evidence is conflicting. Causal importance cannot be ascribed to disturbances of any one gland, though in certain cases a general glandular imbalance may be a predisposing factor. The changes of senility are undoubtedly important, but these are not imitated in their entirety by castration or by the premature menopause which only occasionally gives rise to changes in intra-ocular tension.

The author repeated the work of Passow and Wesseley on the calcium-potassium equilibrium in the blood of patients with glaucoma. He confirmed the presence of a fairly high calcium level in most cases, but found that the potassium varied above and below the values considered normal. The blood picture that he found showed only a slight increase in granulocytes with a decrease in blood platelets.

The influence of the neurovegetative system on the eye has been studied in Italy, especially following the work of Angelucci and his school. It seems to be established that patients with glaucoma are unstable in their neurovegetative reactions, but this instability does not correspond to a uniform vagotonia, as was once supposed. It is rather an absence of the normal balance between the sympathetic and parasympathetic systems, which varies toward one or the other system at different times.

The importance of changes in the blood vessels is emphasized, as it is perhaps through such changes that senility, syphilis, nevus flammeus and other conditions affect the intra-ocular tension. The author investigated the vascular system in a series of thirty-two cases and found lesions of the veins, such as hemorrhoids, angiomas, varices and dilatation of the facial veins, with high venous pressure in a large proportion of cases. Anomalies and changes in the eye itself, such as hyperopia and hardening of the sclera in old age due to the deposit of fat and calcium, are evaluated as predisposing causes.

The author concludes that no single factor may be considered the important cause of glaucoma, but that the sum of the hereditary and acquired changes mentioned makes up an individual constitution in which glaucoma develops under the stimulus of an acute toxemia or infection.

S. R. GIFFORD.

THE EFFECT OF HYPERTONIC SALT SOLUTIONS INJECTED SUBCONJUNCTIVALLY IN GLAUCOMA. I. JOHN, Arch. f. Ophth. **126:** 592. 1931.

After instillation of 2 per cent holocaine, from 0.3 to 0.5 cc. of 10 per cent of sodium chloride was injected subconjunctivally, after which the tension in eyes with normal pressure went up from 7 to 13 mm. and returned to the original level within eighty minutes (with one excep-

tion). The procedure was quite painful. Cases of secondary and of chronic inflammatory glaucoma responded with a further rise in tension to the salt injection. In cases of simple glaucoma a primary drop in tension seemed to be quite characteristic. This suggests a principle difference between the mechanisms of the inflammatory and the noninflammatory type of glaucoma.

P. C. KRONFELD.

DAILY VARIATION OF TENSION IN GLAUCOMA. A. S. WEISS, Russk. j. opht. **14:** 93 (Aug.) 1931.

The author measured the tension of glaucomatous eyes and that of normal eyes. There were fifty-three eyes of thirty-eight glaucomatous patients. The tension was taken three times daily for from one to seven days. Thirty-one of these eyes were affected with chronic inflammatory glaucoma; eighteen suffered from absolute glaucoma and three from chronic simple glaucoma; five were in the prodromal stage, and three eyes had acute inflammatory glaucoma.

The only group in which the regular periodicity of variation, i. e., increase of tension in the morning with decrease of it toward the evening, was noticed was in patients in the prodromal stage. The average amplitude of variation in that group was 4 mm. Less regular variation, the majority of cases showing the morning increase of tension, was observed in acute inflammatory glaucoma. The other groups retained this regularity in from only 25 to 45 per cent of the cases.

The author believes that the amplitude of the variation of tension is in direct proportion to the tension, or the higher the tension, the larger the amplitude. The careful observation of the daily variation in tension is important in making an early diagnosis of glaucoma.

O. SITCHEVSKA.

## Injuries

A RARE CASE OF MIGRATION OF A FOREIGN BODY IN THE VITREOUS. LAMPERT and LAMBERT, Arch. d'opht. **49:** 708 (Oct.) 1931.

The case reported concerns a young man who received a penetrating wound of the cornea and lens of his right eye while hammering a piece of bronze. Shortly after the accident he was seen by Rollet, who found a small foreign body in the vitreous with the ophthalmoscope which did not react to the giant magnet. It was localized by roentgen examination. The patient was free from symptoms for two years, when this eye suddenly became painfully inflamed without any prodromal symptoms. The authors, who saw him at this time, observed a prismatic brownish-yellowish mass enmeshed in the iris in the lower angle of the anterior chamber between 6 and 7 o'clock. This they removed with some difficulty with forceps through an incision at the limbus. The eye subsequently became quiet. They suggest as possible factors the normal physiologic movements of the eye, osmotic currents and the effects of an inflammatory reaction about the foreign body.

S. B. MARLOW.

## Instruments

A New, Handy and Inexpensive Electrical Ophthalmoscope. R. Korbsch, München. med. Wchnschr. **79:** 267 (Feb. 12) 1932.

Two years ago the author described a combination electrical instrument for use in work on the ear, nose and throat. The ophthalmoscope presented is to be used in connection with this same set-up of battery and resistance. The price is from one-half to one-fifth that of the usual electric battery-handle instruments, and it has the advantage of focusing the light element instead of simply reflecting it by a right-angled prism. It consists essentially of a conical tube 3 cm. long, having a diameter of 10 mm. at one end and of 5 mm. at the other. A tiny lamp (*Mignonlampschen*) is the source of light. The instrument may be held directly before the eye with the 13 diopter magnifying lens which is fastened in its bracket above the illuminating element used to view the fundus. No auxiliary lenses are used.

L. L. Mayer.

## Lacrimal Apparatus

A Case of Bloody Tears Due to Commotional Disturbance of the Vegetative Nervous System. Bolotte and Fribourg-Blanc, Arch. d'opht. **48:** 697 (Oct.) 1931.

The authors first describe a case of recurrent conjunctival hemorrhage producing bloody tears following a cranial traumatism which seemed to have upset the vagosympathetic equilibrium. The production of bloody tears seemed to be associated with attacks of conjunctival hyperemia due to a functional disturbance of the craniofacial sympathetic nerve. They discuss separately the three chief symptoms of their patient, occipital headache, loss of vision of the right eye and repeated conjunctival hemorrhages.

Bloody tears have been noted a number of times in young subjects, especially in women. They are not due to a real hemorrhage but to rupture of conjunctival capillaries, the contents of which mingle with the tears. In 1923, Gabriélidès reviewed this condition and collected the reports of eighteen authors from the literature, the majority describing only one case. Frequently bloody tears have occurred at the menses. Attention is called to the similarity of these cases to epistaxis, and the variety of evidence of endocrine and vasomotor imbalance associated with them is pointed out. The occipital headache the authors feel was due to increased intracranial pressure, caused by an increased secretion of cerebrospinal fluid as the result, possibly, of damage to a sympathetic plexus in the choroid plexus. They explain the loss of vision and its subsequent improvement by direct damage to the optic nerve.

While the headache and visual defect cleared quite rapidly, the vasomotor disturbance persisted for a long time. Six months after its onset, the authors did not feel justified in saying that it would not recur.

S. B. Marlow.

BOTH LACRIMAL SACS LINED WITH STRATIFIED PAVEMENT EPITHELIUM: REPORT OF A CASE. M. BOEMER, Klin. Monatsbl. f. Augenh. **87:** 198 (Aug.) 1931.

A man, aged 50, received treatments for nasal lupus from 1911 to 1926. After destruction of the cartilaginous portion, the lupus finally healed under applications of quartz light and x-rays. The man was admitted to the eye clinic of the University of Tübingen in 1930 with a large serpent ulcer and hypopyon in his right eye. Abundant pus, containing pneumococci, could be pressed out of both lacrimal sacs, which were extirpated. They were lined with many layers of stratified pavement epithelium. The walls of the sacs were slightly thickened; their interior covering was furrowed. No pus or bacteria were found in them. Similar metaplasia of the epithelium of the conjunctiva has been observed in cases of chronic conjunctivitis. Boemer found no evidence of tuberculosis or lupus of the sacs; he suspected that the pavement epithelium of the canaliculi proliferated into the sacs, replacing the mucous lining which had been destroyed by the dacryocystitis. A few related conditions have been described by McKee (*Tr. Am. Ophth. Soc.* **23:** 54, 1925).

K. L. STOLL.

## Lens

A CONTRIBUTION TO THE STUDY OF EXFOLIATION OF THE LENS CAPSULE OR GLAUCOMA CAPSULO-CUTICULARE WITH ANATOMICAL PREPARATIONS. M. SOBHY BEY, Brit. J. Ophth. **16:** 64 (Feb.) 1932.

After pointing out that similar conditions of the pupillary border were noted in the prebiomicroscopic era but were differently interpreted, as they were always ascribed to a bleaching of the pars iridica retinae in old people as a senile change in chronic glaucoma due to increased intraocular pressure, or, in diabetes, to depigmentation of the pigment epithelium of the iris—the author analyzes the literature and then gives his own personal observations, consisting of data in twenty-four cases. In two of his cases he describes the histopathology. He summarizes as follows:

| | | |
|---|---|---|
| Eyes showing pupillary masses | 41 of 44 = 90 | per cent |
| Eyes with rise of tension | 29 of 44 = 65.8 | per cent |
| Persons showing glaucoma | 13 of 24 = 54 | per cent |
| Cataractous eyes | 17 of 44 = 39 | per cent |
| Pigment on posterior surface of cornea | 6 eyes of 44 = 13.6 | per cent |
| Youngest age | 55 years | |

Total: 24 patients = 48 globes, 44 of which were used in the statistics, 4 being destroyed by other diseases.

This ailment is insidious and of long duration and takes years for its development. It is rare, but the more it is looked for the more it is met with. According to the Zurich Eye Clinic, seventeen per thousand of old people above 60 years of age (not hospital patients) show Vogt's syndrome. Half of this number at least, if not all, tend sooner or later to develop chronic glaucoma, and it figures as 8.6 per cent of chronic glaucoma. This disease is not an inflammatory one; it is a senile degeneration of the lens capsule of some sort. Depigmentation of the iris tis-

sues plays an important part in this process. As a rule, the tension rises gradually, and it is higher in those eyes that show degenerative process most. The intra-ocular pressure, before passing to the abnormal, may stay for a long period within the normal limits, but is decidedly higher than in eyes that do not show the condition at all, or only to a slight extent. This condition by itself is apt to have a deleterious effect on the field of vision and the optic nerve.

The edge of the pupil undergoes certain definite changes; soon after the exfoliation of the lens capsule in early cases, isolated felt masses, bluish white, begin to appear, principally at the pupillary border, on, or within, the corrugated ring of the retinal pigmented cells of the iris (pigment cells of the back surface). Later on, this corrugated black or darkly pigmented ring at the edge of the pupil disappears slowly, either partially or totally. A gray band or ring replaces the normal black one. This abnormal appearance of the pupillary border can be produced either by the superposition of the felt masses at the pupillary border or by ectropion of that border showing the aforementioned newly formed membrane, and produced by the felt masses on the back of the iris.

These facts are observed in the first eyeball submitted for histologic examination. From the second globe submitted to the same process, it was learned that a similar gray ring can be produced by the depigmentation of the very tip of the iris tissues, bringing to view the proximal edge of the sphincter muscle.

The article is illustrated with a colored plate and numerous photomicrographs.

W. ZENTMAYER.

TREATMENT OF TRAUMATIC CATARACT. F. WIBAUT and L. K. WOLFF, Klin. Monatsbl. f. Augenh. **87:** 49 (July) 1931.

The substance of the lens consists mainly of gel, a hydrophil colloid which, in the same manner as gelatin or agar, will swell as soon as water or diluted acid is added. Experiments in animals have shown that the rupture of the lens capsule will allow this process, although temporary or permanent closure of the wound in the capsule by fibrin will prevent the spread of swelling. This experiment prompted the idea of replacing the aqueous humor temporarily with a gas. Air injected for this purpose into the anterior chamber was of no avail because its oxygen was rapidly absorbed. Nitrogen, employed in analogy with the experiences in artificial pneumothorax yielded better results. After preceding experiments on animals, air was used in one human eye, and nitrogen in the eye of another patient, both of whom had injuries of the anterior lens capsule. The process may be tried in cases in which the anterior capsule of a restless patient has been injured during an operation for glaucoma. The nitrogen need remain in the anterior chamber only long enough to allow closure of the wound in the anterior capsule; it will be absorbed within forty-eight hours.

K. L. STOLL.

CATARACT FORMATION IN MONGOLIAN IDIOCY. A. ELSCHNIG, Med. Klinik **28:** 217 (Feb. 12) 1932.

The author reviews the literature on the condition, which dates back only two decades. The prevailing impression is that it is associated with an endocrine disorder, and improvement in the general condition is

expected and usually shown under thyroid medication. The author reports two cases, giving a detailed description of the ocular and general findings. He makes a plea for early operation (during the first week of life in the congenital type) on the cataractous eyes of these patients, in order to prevent amblyopia.

L. L. MAYER.

## Methods of Examination

THE BIOMICROSCOPY OF TRACHOMATOUS PANNUS AT ITS ONSET. CUÉNOD and NATAF, Arch. d'opht. **48: 737** (Nov.) 1931.

The biomicroscopy of trachoma in general and of trachomatous pannus in particular has, as yet, been too little studied, and has failed, in the authors' opinion, up to the present, to be sufficiently extended, although it has already been written about by several different observers, especially Gaellmaertz and Morax and Petit. In the experience of the authors, slit-lamp examination not only of the tarsal conjunctiva but also of the cornea has real value. With it the earliest signs of corneal infiltration can be found. This infiltration begins by an exaggeration of the lymphoid tissue which appears to be arranged on the one hand like a brush, the fibers of which run with the capillaries, and on the other in small masses which undoubtedly act as pathologic "spines" and attract and favor vascularization in their immediate vicinity. The changes take place among the normal capillary network of the limbus above and give an appearance at first which suggests that the epithelium of the cornea is less transparent, as though thickened and elevated by small wedges which are not more than 2 mm. in diameter and produce a punctate appearance. This opacity diminishes toward the middle of the cornea and is absent in the lower portion. Under high magnification the spaces between the capillaries appear as whitish masses in which more dense white clumps stand out. Observation during the development of this opacity suggests that the lymphatic infiltration appears before the vascularization.

S. B. MARLOW.

## Neurology

THE OPHTHALMOLOGIC FUTURE IN CASES OF TUMORS IN THE REGION OF THE HYPOPHYSIS IN WHICH OPERATION HAS BEEN PERFORMED. MARTEL, MONBRUN and GUILLAUME, Arch. d'opht. **48: 529** (Aug.) 1931.

The authors believe that the pallor of the optic disk, commonly regarded as optic atrophy, which is seen in cases of pituitary tumor, is not a direct atrophy of the nerve fibers but is intimately related to the ischemia due to the compression of the vascular supply to the chiasm and optic nerves by the tumor. They point out that the term optic atrophy implies a degenerative lesion not capable of recuperation with good function—a condition which obtains when operative interference is resorted to too late. A study of their cases indicates that the lowered visual acuity and contracted field are not permanent, even though the disks present the typical appearance of atrophy. After operation, the whiteness of disks disappears, vision improves after some weeks, sometimes to normal, and the field enlarges. Even in those cases in which the optic

nerve is seen at operation to be reduced to a fine thread, the blood supply becomes reestablished, but restoration of function is not to be expected.

The authors review the blood supply to the hypophysis, chiasm and optic nerves and present their reasons for believing that it is interference with the circulation that produces the changes in the optic nerve. They have never seen the peculiar yellowish atrophy described by Benedict, and Cushing and Walker as typical of this condition.

Four cases are described in which vision became normal in one eye at least after operation. The authors attribute the improvement to the fact that the intervention was done early. A fifth more advanced case is also described in which only partial visual improvement resulted.

The surgical treatment offers new hope for these cases. Medical treatment and the use of roentgen rays do not prevent ultimate blindness nearly so certainly, in the author's opinion. S. B. MARLOW.

TRAUMATIC DIABETES INSIPIDUS CAUSED BY LESIONS IN THE REGION OF THE HYPOPHYSIS. J. WOLFF, Klin. Monatsbl. f. Augenh. **86:** 799 (June) 1931.

During a fight, a small wooden cone, about 13 cm. long and 1.5 cm. in diameter, was hurled into the left eye of a man, aged 37, in such a manner that only 2 cm. protruded between the closed and edematous lids. Cerebral symptoms and hemorrhages from the mouth and orbit followed. Several blood vessels had to be clamped and tampons inserted. The left globe was pushed toward the temporal side, but was intact, except for the indistinctness of the margin of the disk and the absence of light perception. Both pupils were rigid, and total ptosis and immobility of the right, uninjured eye were observed on the following day. Polyuria with specific gravity of from 1.002 to 1.005 developed after two weeks. The throat, nose and ears presented no pathologic changes; the roentgenograms, however, showed destruction of the ethmoidal cells, a shadowed left frontal sinus and an oblique fracture of the left dorsum sellae, causing lesion of the hypophysis and middle brain system. Both optic nerves became atrophic, causing amaurosis. The third, fourth and sixth nerves and fibers of the sympathetic were destroyed on the right side by splinters of bone. The diabetes disappeared. K. L. STOLL.

ABSCESS OF THE BRAIN AFTER LESION OF THE SKULL AND CHOKED DISK: REPORT OF A CASE. I. TĂNĂSESCU and D. LĂZĂRESCU, Klin. Monatsbl. f. Augenh. **87:** 108 (July) 1931.

Uhthoff found papillitis in 52.9 per cent of brain tumors, but only in 23 per cent of brain abscesses. This is due, according to Eagleton, to softening of the brain substance in abscesses of the brain, where, for the same reason, only slight increase of the lumbar pressure is present. The mere presence of papillitis does not allow any conclusions as to the presence of a tumor or of an abscess in the brain.

The case reported by the authors shows the importance of the ophthalmoscopic findings in connection with an increase and decrease of the cerebral symptoms in an abscess of the brain. A boy, aged 15, was admitted to the hospital six weeks after he had fallen from a horse which, at the same time, kicked the left side of his head. He had right-sided hemiparesis, severe headache, nausea, transient obscurations,

which returned every three to four days, faulty vision of 2/3 in the right eye and 1/6 in the left, and choked disks. A suppurative wound and a fracture of the skull, depressed 5 cm., were found in the left parietal region. The field of vision was concentrically contracted to from 30 to 40 degrees in the right eye, and to from 15 to 35 degrees in the left. An opening of the wound and escape of a large quantity of thick, yellow pus brought no change. Lumbar puncture succeeded in causing cessation of the obscurations; the spinal pressure was normal. The papillitis, however, increased to 2 mm. in the left eye; vision decreased 1/8 and 1/10, respectively, and the fields of both eyes became more contracted, especially the field of the injured side. Trephining was finally performed at the site of the wound, with removal of a large sequester and of about 150 cc. of pus, in which streptococci were found. Recovery followed promptly; the papillitis receded, and vision returned slowly. Half a year later the choked disk had completely disappeared; vision and field of vision again became normal.

Similar observations were made in infected gunshot wounds of the skull during the war (von Szily, A.: Atlas der Kriegsaugenheilkunde, Stuttgart, F. Enke, 1918. von Hippel, E., in Schjerning: Handbuch der ärtzlichen Erfahrungen im Weltkrieg, 1914 to 1918, Leipzig, J. A. Barth, 1922).

K. L. STOLL.

## Ocular Muscles

END-RESULTS IN MONOCULAR ESOTROPIA. L. C. PETER, Brit. J. Ophth. **15:** 642 (Nov.) 1931.

The author believes with a minority of operators that the ideal goal of recovery from squint with single binocular vision can be obtained in the vast majority of young squinters, if systematic measures are instituted, if the squint is treated at the proper age and if the cooperation of the parents can be obtained.

Ideal results include: first, a good visual acuity in the squinting eye; second, single binocular vision which will stand the proper tests; third, good convergence for sustained close work, and fourth, comfort for the patient, which is necessarily predicated on a good muscle balance after treatment is completed. Aside from neglect on the part of parents, the two technical conditions which stand as a barrier to a perfect outcome are amblyopia ex anopsia and an undeveloped or absent fusion faculty. Factors which favor ideal results are: refraction; prevention and correction of amblyopia ex anopsia; training of the fusion faculty; operative intervention.

The basic principles to be observed in operating are:

1. The converging power of the internal recti should be preserved. As a natural corollary, if it is necessary to operate on one or both internal recti, nothing but muscle recession should be practiced. Tenotomy is contraindicated.

2. If the squint can be corrected by shortening one or both external recti, the results will be most satisfactory and normal convergence will not be placed in jeopardy.

3. Supplementary operations, if necessary, should be reserved for the internal recti. There are occasional exceptions to this rule when the internus is definitely in a state of contracture.

4. Only such operations should be selected as will admit of the most accurate correction of the squint.

5. Although the cosmetic appearance of the bulbar conjunctiva is secondary in importance to the accuracy of the restoration of parallelism, it is worthy of serious consideration in the choice of operations.

W. ZENTMAYER.

## Operations

OPTICAL IRIDECTOMY SHOULD BE AN EXCEPTIONAL OPERATION. ROCHE, Arch. d'opht. **48:** 825 (Dec.) 1931.

The author points out that this operation is not without disadvantages and may convert a curable infirmity into an incurable one. This happens as the result of surprises which are possible in the clearing of corneal opacities. He cites two cases that illustrate his point very well. He raises the question as to whether this operation is advisable in all cases in the presence of a permanent scar, even when vision is improved after dilating the pupil. He cites a case in which the patient was not only not improved but actually made more uncomfortable because of dazzling, etc. He calls to mind Terrien's statement in his textbook of ophthalmology: "Optical iridectomy is an uncertain measure, and may only have the result, if the iridectomy is too large, of giving rise to dazzling." To this Roche would add that its use sometimes risks converting into an incurable infirmity a condition that would have cleared spontaneously.

S. B. MARLOW.

## Physiology

THE CEREBRAL CIRCULATION: XIV. THE RESPIRATORY QUOTIENT OF THE BRAIN AND OF THE EXTREMITIES IN MAN. W. G. LENNOX and ERNA LEONHARDT, Arch. Neurol. & Psychiat. **26:** 719 (Oct.) 1931.

A comparison is made of the oxygen and carbon dioxide content of arterial blood going to the brain and venous blood coming from the brain (internal jugular vein). From this the respiratory quotient of the brain can be obtained. Lennox finds it equals 0.95, which suggests that the metabolism of nerve tissues involves a larger use of carbohydrates than is the case with muscle. The concentration of sugar in these vessels was then determined, and it was found that more sugar disappears from the arterial blood in passing through the brain than in passing through large masses of muscle, as an arm or a leg. This indicates that the metabolism of the brain is largely concerned with the use of dextrose.

F. H. ADLER.

THE CEREBRAL CIRCULATION: XVII. CEREBRAL BLOOD FLOW AND THE VASOMOTOR RESPONSE OF THE MINUTE VESSELS OF THE HUMAN BRAIN TO HISTAMINE. SOMA WEISS and W. G. LENNOX, Arch. Neurol. & Psychiat. **26:** 737 (Oct.) 1931.

It has been shown previously (Weiss, Ellis and Robb: *Am. J. Physiol.* **90:** 551, 1929) that dilatation of the minute cerebral blood vessels of man occurs with injections of histamine. The present paper deals with a confirmation of this by estimating the increased flow of blood through the brain.

F. H. ADLER.

## Retina and Optic Nerve

EXPERIENCES WITH THE IGNIPUNCTURE. N. BLATT, Arch. f. Ophth. **127:** 137, 1931.

The author reports eight cases of retinal detachment, three of which were cured by one or several ignipunctures. He summarizes the present status of the ignipuncture problem without adding anything new. The following table contains the number of cured cases as observed by the various authors:

| Author | Number of Cases Operated On | Percentage of Cured Cases |
|---|---|---|
| Lindner | 25 | 44 |
| Gonin | 81 | 53 |
| Brueckner | 12 | 41 |
| Vogt | 26 | 42 |
| Elschnig | 14 | 64 |
| Arruga | 33 | 50 |
| Stock | 22 | 36 |
| Clausen | 20 | 25 |

P. C. KRONFELD.

ORIGIN AND TREATMENT OF IDIOPATHIC AND TRAUMATIC RETINAL DETACHMENT. K. LINDNER, Arch. f. Ophth. **127: 177**, 1931.

After a few historical remarks, Lindner discusses first the rôle played by the retinal tears. It has not yet been proved scientifically that the retinal tears are the cause of the detachment, but from clinical observations and experiments with a schematic eye, it seems to be a highly probable assumption. With increasing ophthalmoscopic skill and experience, retinal tears are now found almost regularly in fresh cases of detachment. Among sixty-nine cases of detachment in which treatment was given at the second eye clinic in Vienna during 1930, fifty-five showed one or several retinal holes (79.5 per cent). In the twelve cases in which tears could not be found the ophthalmoscopy was difficult because of cloudy media or other reasons. A total detachment makes the search for tears extremely difficult because of the larger surface to be searched and because of the many folds behind which tears can hide. For the same reason it is more difficult to find defects in old detachments than it is in recent cases.

In a large percentage of cases more than one tear is found. Among the fifty-five cases mentioned thirty-one had only one, thirteen had two, two had three and nine had more than three holes before the first operation, counting holes which lay so close to each other that one cautery took care of all of them as one hole. In the thirty-one cases in which only one tear was seen before the first operation, the postoperative course suggested the presence of a second tear in at least ten cases. Many of these tears found after surgical procedures may be the result of the operation.

As to the location of the tears, Lindner confirms Leber's and Gonin's statement that they occur most frequently in the upper temporal quadrant; next in frequency comes the lower temporal quadrant and then the upper nasal quadrant. Thirty-one of Lindner's cases showed tears in the temporal, and fourteen in the nasal half of the fundus. Gonin found the same relation (46:20) in his material.

Among seventy patients with retinal detachment, nine had a myopia of from 1 to 5 diopters, twenty-two of from 5 to 10 diopters and twenty-six more than 10 diopters. Thirteen had emmetropia or hypermetropia. Six eyes in the whole group were aphakic.

Cases with anatomically or clinically visible tears without detachment have been observed, but do not speak against Gonin's theory of the etiologic rôle of the tear, because in every one of these cases it could be shown that there were firm chorioretinal adhesions in the neighborhood of the tear.

On a number of cases Lindner shows that the closure of all retinal defects present at the time of the operation in itself and not any of the secondary effects of the operation leads to complete reattachment.

Lindner believes that the mechanism of retinal detachment depends a great deal on the condition of the vitreous. In an eye with normal vitreous, the process is as follows: An injury or a preexisting pathologic process produces a retinal hole after which the vitreous adjacent to the hole is liquefied, and through the movements of the eye—this is illustrated on a model—some fluid is forced behind the retina. The choroid easily absorbs this fluid which, however, is instantly replaced by vitreous fluid and later by secretion of the ciliary body. Under the influence of this abnormal secretion, the vitreous shrinks down to a smaller volume, and the classic picture of a tightly stretched retinal vesicle with a tear on its prominent point becomes visible. A larger choroidal area has been exposed in the meantime, and the ciliary body is asked to produce more fluid of abnormal nature, which most likely on its way backward "densifies" the vitreous. Within a few weeks the retinal vesicle seems more relaxed, its surface shows folds and wrinkles and is easily movable. The vitreous has been more or less liquefied and does not pull on the retina any more.

If the whole process of the formation or of the secondary enlargement of the tear takes place rapidly, then the exposed choroid removes more fluid than the ciliary body can possibly produce, and acute hypotony sets in. Lindner is aware of the fact that the condensation of the vitreous, which is an essential factor in his theory, has not been demonstrated experimentally.

In cases with fluid vitreous retinal detachments take a different course. The inertia of the eye in connection with its movements plays a dominating rôle, and by putting the eye at rest very extensive temporary reattachments can be accomplished. To eliminate the action of these mechanical forces, Lindner makes his patients wear a pinhole.

Lindner used Gonin's method until July, 1930. His results during the first half of 1930 were much worse than those obtained during 1929, which he explains by differences in the type of detachments and secondary factors. He also became more familiar with the injurious effects of the ignipuncture, namely, necrosis of the retina around the burned area and formation of secondary holes centrally to the cauterized areas. Since July, 1930, Linder and his associate, Guist, have been using a new method which consists of surrounding the retinal defects by multiple chorioretinal adhesions produced by cauterization of the exposed choroid with a dry potassium hydroxide stick. To expose the choroid, small holes are drilled in the sclera with a 1.7 mm. trephine. The accurate localization of the defect is not necessary any more. Areas in which tears

are suspected but not definitely seen are treated in the same way. If the tear is located in front of the equator, it is surrounded by a row of trephine holes which starts out from and goes back to the ora serrata. The new method is very difficult, but does not lead to shortening or shrinking of the retina, which seems to be the main disadvantage of the ignipuncture. The results obtained with the new method will be published by Guist.

P. C. KRONFELD.

PROGRESSIVE ATROPHY OF THE CHOROID WITH PIGMENT DEGENERATION OF THE RETINA. REPORT OF CASES. F. WERKLE, Klin. Monatsbl. f. Augenh. **87**: 173 (Aug.) 1931.

Fuchs, in 1896, described atrophia gyrata choroideae et retinae as a disease related to retinitis pigmentosa. Werkle's present contribution furnishes a description and colored pictures of four successive stages of this rare condition. The patient belonged to a family in which thirteen of thirty-three members suffered from hereditary hemeralopia. Of the six children of the first generation only the three daughters were afflicted, while three sons were healthy. In the second generation a daughter's sons inherited the disease. Both sexes were afflicted in the third generation. Clinical examinations of six of the thirteen persons were made; some disparity in the fundus with that of typical pigmented retinitis could be observed, but none of the patients showed typical aberrations of the disks and retinal vessels. Two brothers of the third generation, aged 10 and 20, showed the most striking changes in the fundus, representing different stages of progressive atrophy of the choroid, except in the region of the macula. Both boys were perfectly healthy and free from any anomalies of development. A third patient, aged 50, and not related to this family, presented similar symptoms, and also an etiology of heredity. In this case the fundus showed an almost glaring white reflex, except in the periphery, close to the ora serrata, while the area of the macula appeared as a dark red circle. The optic disk was faintly pink. The patient had extensive contractions of the field of vision, in addition to the hemeralopia. All three patients represented a typical form of a slowly progressing degeneration of the retina with atrophy of the pigment epithelium and of the choroid. Total atrophy of the retina with preservation of the macula must be considered as terminal stages of Fuchs' atrophia gyrata. The primary cause of this disease has been attributed by some to primary hypoplasia of the retinal vessels, and by others to deficient development of the nervous elements of the retina. The primary lesion, in Werkle's opinion, is located in the choroid, as indicated by the ophthalmoscopic findings and the defects of the fields of vision, which by their ring-shaped arrangement correspond exactly with the atrophic foci in the fundus. The relative intactness of the vascular system of the retina seems to exclude its primary lesion as the cause of the atrophia gyrata. The fact that the process begins in the choriocapillary layers renders it probable that the choroid was inferior even in its germ cells. Especially dense meshes of the capillaries of the choroid, found in the macular region, help to explain its preservation. Leber's term, "degeneration of the pigment of the retina with extensive atrophy of the choroid," or better, "progressive atrophy of the choroid with degeneration of the pigment of the retina," the author's term, seems to be more correct than that of Fuchs. The condition is not

identical with Oguchi's disease. Progressive myopia cannot be responsible etiologically, although high degrees of myopia have been frequently observed in progressive atrophy with degeneration of the retinal pigment. The progressive nature is indicated by increasing hemeralopia, by disturbances of the field of vision and by an increase of the changes in the fundus. Therapy is of no avail; the prognosis depends on the degree of the changes and the rapidity of the progress. Total atrophy does not result in every case.

K. L. STOLL.

RETINITIS COMBINED WITH TUMOR OF THE CEREBELLUM (SO-CALLED LINDAU OR VON HIPPEL'S DISEASE) AND ITS RELATION TO COATS' DISEASE. C. OGUCHI, Acta Jap. Ophth. Soc., June, 1931, p. 91.

Our conception of von Hippel's disease has been enlarged after its association with tumors of the cerebellum was determined by Lindau. The nature of the process is still uncertain, and there are two opinions concerning it: (1) that it is angiomatous, according to von Hippel, Lindau and others, and (2) gliosis, gliomatosis or glioma, according to Meller, etc.

Coats' disease is not well understood. There is no similarity in the anatomic description of the two conditions (von Hippel's and Coats'). In the beginning stage, dilatation and nodes in the retinal vessels are observed by some, while others speak of a diffuse, glistening opacity of the fundus.

The author's patient was a boy 10 years of age, who exhibited in February, 1930, a peculiarity in his gait. Then followed visual disturbances, headache and vomiting, and in July, he was totally blind. In September, he complained of loss of hearing, and hydrocephalus was present. On admission to the surgical clinic of Professor Saito, the child showed Coats' disease, type 1, hydrocephalus, disturbances of hearing, right-sided facial paralysis, restless choreic twitchings and high fever. On October 12 death occurred following convulsions.

Autopsy showed a cerebellar tumor of the right pontine angle and a broad adhesion between the pia and the dura, with dilatation of all of the ventricles. The tumor pressed the medulla toward the left, and as it had no wall it was regarded as a false cyst. Posteriorly, the tumor became more vascular without definite angiomatous or cystic formation. There were numerous hemorrhages in the surrounding areas into which the tumor cells had extended in the form of cell groups. Fat-containing cells were everywhere present.

The left eyeball and posterior half of the right eyeball and the optic nerves were removed. Examination showed marked hypertrophy of the glia fibers with increase of the glia cells in the nerve fiber layer and in the papilla, as well as enormous hypertrophy of Müller's fibers, with infiltration into the external layer. Exudation had taken place in the subretinal space and on the inner surfaces of the retina, which contained numerous broken-down glia and fat granular cells. Newly formed and dilated blood vessels were visible in the retina near the disk. There was enormous infiltration of cells about the vessels. Glial and connective tissue hypertrophies were present in the subretinal spaces and joined the choroid. There was a projecting gliomatous tumor in the ciliary body; glial and connective tissue structure occurred at the disk. The retina

was in folds with marked fibrous formation of newly formed connective tissue. In the optic nerve there was a dilatation of the intervaginal space with occlusion of this space with gliomatous tissue. The gliomatous changes were more marked than the angiomatous in the retina and in the cerebellar tumor.

The case, on the whole, represented a malignant glioma in Smirnoff's sense. The process was a rapid one.

According to Oguchi, the conception of Lindau's disease should be enlarged. There are on one side angiomatous tumors in the brain and in the retina, which have a chronic course, and on the other, more rapid processes, rather of the malignant gliomatous type. It is difficult to decide whether the origin of these cells is ectodermal or mesodermal. Microglia, and especially Hortega cells, are now regarded as mesodermal.

It is difficult to differentiate between Coats' and von Hippel's disease. If the newly formed cells are very toxic, exudation occurs in the retina and Coats' disease develops, but if dilatation of the vessels and connective tissue preponderate, the disease is known as von Hippel's. It would probably be better to have Lindau's disease include angiomas of the brain and the retina, gliomatosis of these structures and the intermediate type (angiogliomatosis).

A. KNAPP.

## Trachoma

THE MECHANO-VACCINO-THERAPY OF TRACHOMA AND ITS COMPLICATIONS. C. ADDARIO, Ann. di ottal. e clin. ocul. **59:** 600 (July); 675 (Aug.) 1931.

The author reviews recent work on the cause of trachoma, including the successful human inoculation carried out by the elder Addario, which has convinced him of the etiologic importance of B. granulosis. A vaccine was made from the Rockefeller strain of this organism by Professor Manfredo of Palermo, and was used in treating patients by the author. In the form of an ointment, the vaccine was placed in the conjunctival sac twice daily. Four types of vaccine were made, with which over 400 patients were treated. With the more concentrated vaccine, containing from 5 to 10 per cent of bacterial substance, an acute conjunctival inflammation was produced, which lasted from four to five days, and was followed by relief of the previously existing inflammatory symptoms. The lesions themselves, however, showed little effect when the vaccine was used in this way alone. Cases with pannus and superficial keratitis showed a marked improvement in these conditions.

The author believes that the changes in trachoma are of two kinds, the specific granulomas, which are small, transparent, tubercle-like nodules, and the secondary changes of chronic inflammation which surround them. An effective treatment must expose the specific lesions to the therapeutic agent, and stir up the inflammation into an acute one, with active phagocytosis and absorption of the degenerated tissue. These conjunctival lesions are the cause of keratitis, which will recur unless they are cleared up.

Hence in a further series of cases, a combination of abrasion or expression of the follicles followed by use of the vaccine was tried. One eye served as a control, being treated by vaccine or abrasion alone.

the other being used for the combined method. The results were excellent, all the follicles being absorbed and the conjunctiva returning to normal after from twenty to thirty days. In the eyes treated by either method alone there was little change in the condition. When marked pannus was present, peridectomy was performed, following which an ointment of vaccine was placed in the sac and the eye was bandaged for several days. Marked clearing of the pannus was observed when this was done.

Detailed histories are given of forty-five cases in which the combined method of treatment was used.

The author concludes that the use of the specific vaccine, after crushing or expressing the trachomatous lesions, causes their rapid disappearance and prevents the occurrence of new lesions, which is the rule after mechanical treatment alone. Often after a single expression one or more follicles are left, and these must later be expressed to expose the infected tissue to the action of the vaccine. To prevent recurrence of pannus the granules at the limbus must be thoroughly scarified or curetted and treated afterward with vaccine. S. R. GIFFORD.

## Tumors

A CASE OF PRIMARY MELANO-CARCINOMA OF THE CILIARY BODY. R. H. B. BARROW and H. B. STALLARD, Brit. J. Ophth. **16:** 98 (Feb.) 1932.

The patient, a man, aged 61, presented himself because of defective vision of the right eye (not recorded). Two years previously corrected vision was 6/6.

There was a swelling situated in the iris and in the filtration angle at 6 o'clock. The summit impinged on the posterior surface of the cornea. The greater part of it was of a grayish brown, except in the filtration angle where it had a "pearly" appearance, with two small vessels running over its anterior surface. In this vicinity the anterior ciliary vessels were dilated and engorged. The swelling did not extend to the free margin of the iris. The reaction of the pupil to direct light was sluggish, but the consensual reaction was brisk. The pupil dilated well with homatropine, and it was impossible to detect by clinical methods any neoplasm in the ciliary body. The lens was opaque in the vicinity of the neoplasm and upward to the pupillary area. Tension was normal. There was no evidence of an inflammatory lesion.

The patient was in good health. No neoplasm was found elsewhere in the body. The Wassermann reaction was negative. A diagnosis of sarcoma of the ciliary body and iris was made.

The eye was excised. Situated on the iris and ciliary body at 6 o'clock was a neoplasm 3.5 by 3 mm. The cut surface presented a stippled appearance of white dots on a gray background. With a loupe some areas of myxomatous degeneration were visible.

Histologically, the growth arose from the ciliary epithelium and infiltrated the ciliary body. At the periphery the cells were arranged in rows of short cubes and resembled those of the ciliary epithelium, some pigmented and others nonpigmented. In the center of the neoplasm the cells were disposed irregularly. There were areas of hyaline, colloid and myxomatous degeneration, also cystic spaces lined by pigmented epithe-

lial cells some of which were very swollen. Lymphocytes were present at the advancing edge of the neoplasm, and some were aggregated into large clumps among the tumor cells. Mitosis was evident in some of the cells of the neoplasm. The rest of the iris was infiltrated by the neoplasm, which was pressed against the ligamentum pectinatum and occluding the filtration angle. The canal of Schlemm and the walls of a tributary from the deep scleral plexus of veins were also invaded. At one site the endothelium was stripped from Descemet's membrane and the intervening space filled by tumor cells. In the stroma of the iris some cystic spaces were seen, lined by short cuboidal epithelial cells.

W. ZENTMAYER.

## Therapeutics

THE MANAGEMENT OF TUBERCULOUS DISEASES OF THE EYES AND THEIR ADNEXA, WITH ROENTGEN AND RADIUM RADIATIONS. W. HOFFMANN, Deutsche med. Wchnschr. **57**: 1448 (Aug. 21) 1931.

Radium and the x-rays are usable in ocular conditions just as they are in general tuberculosis when other therapy such as tuberculin gives no benefit or is contraindicated. Through the work of Birch-Hirschfeld we have become acquainted with the possible untoward effects of these radiations. From time to time reports of the use of rays in ocular tuberculosis have appeared in the literature. The author collected thirty-six cases. Thirteen patients were cured with radium and eleven with the x-rays. For treatment of tuberculous conditions of the eye not involving the globe, the author recommends the following procedure for the use of radium: 1. For a small field, a single dose of 20 per cent skin unit is used. The applicator is of 0.2 mm. platinum and 1 mm. copper applied directly for thirty-six seconds (amount used evidently 200 mg.). The dose is 90 mg. hours. 2. For large fields, a 1 cm. skin distance for forty-eight minutes is given over three successive days.

For the x-rays, the author uses a 3 mm. aluminum filter with a 30 cm. skin target distance on from 100 to 130 peak kilovolts, giving a dose of 90 units (roentgen units). This may be repeated after several weeks if necessary, with a maximum of three applications to a series. Stocks' experience with irradiating the eyeball itself would point to a similar dosage. Relief of symptoms in this type of case has been marked and prompt. In conclusion, the author states that it is his firm conviction that tuberculosis of the adnexa of the eye is best treated by radiation. With further reports on its use in diseases of the globe, together with animal experimentation, he believes that use of the x-rays and radium will be shown to be the most efficacious.

L. L. MAYER.

# Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

## COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

*Jan. 21, 1932*

LEIGHTON F. APPLEMAN, M.D., *Chairman*

A CORNEAL DYSTROPHY. DR. J. MILTON GRISCOM.

Dr. Griscom presented a case of dystrophy of the cornea in an Italian woman, aged 31, who applied to the Wills Hospital on Jan. 4, 1932, because of failing vision in the right eye. External examination revealed the presence of numerous small, superficial, discrete, white spots in the pupillary area of the cornea of each eye, unaccompanied by any evidence of irritation. The epithelium was uniformly smooth, and its reflex was unimpaired. There was some loss in corneal sensitivity. There was an area of old choroiditis, and adjacent to this an area of acute exudative choroiditis in the upper outer quadrant of the right fundus, accompanied by many large vitreous opacities. The fundus of the left eye was normal.

Slit-lamp examination of the cornea, made by Dr. Alfred Cowan, was reported as follows: "There are a number of grayish lesions varying in size up to 0.5 mm. in diameter, more or less regularly distributed over the central portion of each cornea, extending over an area about 6 mm. in diameter. The surface of the cornea in specular reflection is seen to be perfectly smooth. By direct illumination it can be seen that most of the lesions consist of a flat granular slightly concave surface with well defined but irregular edges. There are also some dot opacities and some opaque streaks. With a narrow slit, the concave lesions, which are the most common, are seen to lie mostly beneath the epithelium, in Bowman's membrane or just behind it, a few extending back to about the middle of the cornea proper. The dot and streak opacities are mostly in the substantia propria. The cornea between the opaque lesions contains very fine granules, giving the whole a slightly hazy appearance. By retro-illumination the lesions are opaque. Descemet's membrane is intact. There are some brown keratitic precipitates. The lenses are clear, with many brown dot-like vitreous opacities in the right eye."

The case appeared to be similar to the type described by Fehr and allied to the group of corneal dystrophies first described by Groenouw.

DISCUSSION

DR. ALFRED COWAN: In Vogt's new atlas will be found an illustration of a condition almost exactly like Dr. Griscom's case. It is classed among the nodular corneal degenerations and is called

superficial disseminated degeneration. Dr. Griscom brought out in his paper that different types of these corneal dystrophies are nothing more or less than different stages of development of the same process. I think that there is no doubt about this. This is shown in Vogt's atlas by illustrations of four cases in members of one family, two sisters and the only child of each, at the ages of 5, 16, 33 and 42 years. In the child of 5 years there are shown only a few radiating lines in the cornea, not elevated, but just made out. The nodular form of this condition is probably a later stage of development. As a matter of fact, in my experience nodules are the exception rather than the rule—more often streaks, flecks or lines are seen, which are all variations of the same condition. These lesions are not necessarily limited to the anterior portion of the cornea; the condition is slowly progressive. Very rarely, similar lesions have been observed in the posterior as well as in the anterior portions of the cornea.

Dr. William Zentmayer: Has Dr. Cowan noted the condition of the corneal nerves? One observer has stated that they are thickened.

Dr. Cowan: The corneal nerves were not especially noted. I do not consider increased visibility of the corneal nerves particularly significant.

Dr. Zentmayer: In my "Contribution to Ophthalmic Science," together with Dr. Rush, I reported cases seen in three of four generations, of which direct information could be obtained; the great-grandmother was said to have been similarly affected. Bearing on the view that the various types of dystrophy described are all the same, only seen at different stages, there was considerable difference in the ages of the patients when examined, and, presumably, from the statement of the patients, difference also in the length of time they had been affected, but in all the lesions were practically the same.

### Posterior-Lenticonus. Dr. Warren S. Reese.

Dr. Reese said that the bulging of the lens in the patient presented was larger than in the cases he had previously seen, and it contained a large opacity. These two facts probably accounted for the greater diminution of vision in the case presented. The differential diagnosis of true and false lenticonus is not difficult if one has seen such cases previously. False lenticonus is really a variety of nuclear opacity causing an irregular refraction. This is easily demonstrable by noting the distortion of the fundus as one looks through different parts of the lens. The border of a false lenticonus is much less sharp, and the opacity itself is larger. False lenticonus is always seen in older people and causes an increasing myopia. A number of cases have been incorrectly reported as true lenticonus. True lenticonus is rare, whereas the false type is not uncommon. The former is unilateral and nonprogressive, whereas the latter is bilateral and after a period of years causes marked reduction of vision, especially for distance.

#### DISCUSSION

Dr. Alfred Cowan: In eight cases that I have seen, the resemblance was most striking. In every case the process started in the region of the posterior surface of the fetal nucleus, with optically

clear spaces near the posterior Y. There was in each case a stream of opacities, varying in size and degree, which extended backward toward the protuberance. The base of the conus was surrounded by a gray ring. The posterior capsule was intact in every instance, with no irregularity of the double line.

BLOOD-STAINING OF THE CORNEA. DR. WARREN S. REESE.

Dr. Reese presented the case of a child who had been struck in the eye with a strap. Attention was called to the difficulty of differentiating this condition from a lens dislocated into the anterior chamber. This differentiation is particularly difficult in cases of blood staining that have lasted long enough to permit a certain amount of absorption, so that the corneal infiltration assumes the color of a lens.

DISCUSSION

DR. L. F. APPLEMAN: Two patients with blood staining of the cornea recently came under my observation. The first went to the Wills Hospital clinic because of severe pain in the eye from secondary glaucoma which developed after inflammation of the uveal tract. Inspection revealed an exudate in the anterior chamber which had the exact appearance of a dislocated lens. The cornea was clear for a space of 2 or 3 mm. within the limbus all around. The contained mass was lenticular, with the color of a sclerosed lens, and was of about the same size. As a previous operation had been performed, it was thought that on account of some accident the lens was dislocated without deliverance, and that subsequent inflammation had resulted in exudation and interference with filtration, causing the glaucomatous pain from which the patient was suffering. While she was willing for enucleation of the eye, it was deemed advisable to try to get the supposed lens from the anterior chamber, and, failing in that, to perform an iridectomy in the hope of giving her relief.

A keratome incision was made, the point of the instrument penetrating the mass. No lens matter was forthcoming, and with a spatula the supposed lens was found to be a mass of exudate adherent to the cornea in front and to the iris behind. Healing was prompt, and the patient had relief from pain. Before the patient left the hospital, the exudate seemed to be breaking up; when she was seen after an interval of about two weeks, the mass was seen to be disintegrating and much of it had absorbed. I think that the puncture of the mass and the interchange allowed through the iridectomy may have been the cause of the disintegration of the mass.

In reference to the case of blood staining of the cornea in the practice of those more experienced, does the opening of the anterior chamber in these cases in any way hasten the absorption of the exudate?

DR. T. B. HOLLOWAY: I have never been unfortunate enough to open an eye of this character. My experience differs from that of Dr. Reese; I have never been able to see any resemblance between this condition and a dislocated lens in the anterior chamber, despite the fact that reference is not infrequently made to it. The first case of this kind that came under my observation was in a student at the Overbrook School, and I saw another when on duty at the Wills Hospital. About

one year ago I had a patient with traumatism in my ward in whom the anterior chamber was partially filled with blood, and one could conjure up, in this case, a possible suggestion of a partial luxation, but not to the extent of making such a diagnosis.

One of the interesting things about these cases is the chronicity of the staining, as it usually requires months before the cornea becomes clear. Still further, the clearing always begins about the periphery. It is not generally realized that a subconjunctival hemorrhage will also produce blood staining of the cornea in the area adjacent to the hemorrhage, to a degree sufficient to be readily recognized with the loupe. About two years ago I called Dr. Cowan's attention to this fact, and advised him that I had seen it in several cases; in one instance, it was noted when all evidences of the hemorrhage had disappeared.

SARCOMA OF THE CHOROID: A CLINICOPATHOLOGIC CONTRIBUTION. DR. G. E. DE SCHWEINITZ, DR. B. F. BAER, JR., and DR. PERCE DELONG.

From a group of twenty-five cases of sarcoma of the choroid, investigated clinically and pathologically during the last five years, the authors selected seven for description and comment: 1. The value of the ordinary transillumination test was emphasized, the transilluminator being fitted with a small bulb. It was pointed out that a subretinal exudation (not a growth) may completely obstruct transillumination; pupillary transillumination with the aid of the beam of a slit-lamp is highly recommended. 2. Vitreous opacities, as well as considerable disturbance of the vitreous structure, are not infrequently present in cases of retinal detachment due to tumor. 3. A retinal detachment in a hyperopic eye, without a history of injury or the presence of choroiditis and devoid of a retinal tear, should always be regarded with grave suspicion. A *sudden* detachment of the retina with one or more visible rents or dialyses is probably never due to sarcoma. 4. A small circumscribed detachment of the retina in the macular area should almost surely be regarded as an early stage of a tumor. 5. Diminished intra-ocular tension is quite common in sarcoma, not only of the ciliary body, but also of the choroid; in these cases it is probable that the slit-lamp will reveal considerable vitreous degeneration. In short, diminished tension does not necessarily eliminate the diagnosis of tumor. Should the tension, having been low for a considerable time, begin to rise, even for a few points, it is a danger signal and means that the filtration angle is being blocked by exudation, the products of iritis or sarcoma cells. 6. Although localized scleral telangiectasia is most suggestive, it should also be remembered that a patch, especially above, in all respects resembling an ordinary circumscribed episcleritis, may overlie a choroidal or a ciliary body sarcoma. 7. If extrascleral nodules are discovered after enucleation, even though no suspicious fragments are found in the orbital contents by ordinary examination, evisceration of these orbital contents is a proper procedure. 8. Postoperative radiation naturally depends on what is found after the eyeball is removed; certainly after evisceration of the orbital contents it should be employed. 9. The vital prognosis in cases of sarcoma of the choroid, as was shown by statistical

records abroad and elsewhere, is steadily improving when early enucleation is possible, and especially in recent years by the help of properly applied postoperative radiation.

In closing, Dr. Baer said that in the early days irradiation was followed by contraction of the socket, but at the present time he thought that very unusual. In the x-ray department of the University Hospital, the following method is employed for intra-ocular growth: 350 mg. hours, through a 3 mm. lead filter, is given for four doses at intervals of two months. If the growth has broken through, the orbit is infiltrated with 250 mg. hours through a 2 mm. brass filter, for one dose, unless recurrence is observed.

In cases in which there has been breaking through, it is generally the plan to eviscerate the contents of the orbit before subjecting the patient to irradiation, and before operating for intra-ocular tumor, it is wiser to get permission to do a more radical procedure if necessary.

In epibulbar sarcoma the irradiation is given without filtration, 200 millicuries over two areas, one for forty-five and the other for twenty seconds, respectively, and Dr. Baer said that they had three or four cases in which irradiation had been successful in eradicating the growth without surgical intervention.

DISCUSSION

Dr. S. Gross: In addition to the case reported by Dr. de Schweinitz, I should like to refer to one presented before the Section on March 15, 1922. The patient, Mrs. H. D., aged 65, had been operated on by a Philadelphia ophthalmic surgeon for glaucoma. A corneoscleral trephining was performed. Eight months later, on account of continued severe pain, the eye was enucleated, and a tumor was noticed around the nerve at its entrance in the globe. The chief point of interest was that on microscopic examination of the eye, there was found a metastatic sarcoma at the trephined opening. The original tumor was a melanosarcoma of the choroid, about 3.5 by 9 mm., extending outside the globe. The slide was shown to Professor Fuchs, who did not recall having seen one like it.

Histologic Study of the Iris Inclusion Operation in the Eye of the Rabbit. Dr. Edmund B. Spaeth.

This article will be published in full in a later issue of the Archives.

# Directory of Ophthalmologic Societies *

## INTERNATIONAL

### International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217, Faubourg St. Honoré, Paris.
Secretary-General pro tem: Dr. A. Churchill, 66, Boulevard St. Michel, Paris.

### International Ophthalmologic Congress

Secretary: Dr. Franciso Poyales, Olozaga 3, Madrid, Spain.
Place: Madrid, Spain. Time, April 16-22, 1933.

## FOREIGN

### British Medical Association, Section on Ophthalmology

President: Sir J. Herbert Parsons, 54 Queen Anne St., Cavendish Square, London, W. I.
Place: London. Time: July 27-29, 1932.

### Ophthalmological Society of the United Kingdom

President; Mr. A. H. H. Sinclair, 6 Charlotte Square, Edinburgh.
Secretary: Mr. Humphrey Neame, 56 Portland Place, London, W. I.

### Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.

### Royal Society of Medicine, Section of Ophthalmology

President: Dr. E. W. Brewerton, 73 Harley St., W. 1, London.
Secretary: Dr. P. G. Doyne, 60 Queen Anne St., W. 1, London.

### Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.
Place: Paris. Time: July 17-21, 1932.

### German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.

## NATIONAL

### American Medical Association, Section on Ophthalmology

Chairman: Dr. Frederick H. Verhoeff, 82 Commonwealth Ave., Boston.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: Milwaukee. Time: 1933.

### American Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
President-Elect: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Montreal, Canada. Time: September 19-23, 1932.

### American Ophthalmological Society

President: Dr. Edward C. Ellett, 130 Madison Ave., Memphis, Tenn.
Secretary: Dr. Emory Hill, Professional Bldg., Richmond, Va.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

## SECTIONAL

### New England Ophthalmological Society

President: Dr. David W. Wells, Hotel Westminster, Boston.
Secretary-Treasurer: Dr. Theodore L. Terry, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

### Pacific Coast Oto-Ophthalmological Society

President: Dr. W. F. Hoffman, 317 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.

### Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. John H. Harter, 1215 Fourth Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

### Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Sioux City, Iowa. Time: November, 1932.

### Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Birmingham, Ala. Time: November, 1932.

### Southwestern Michigan Triological Society

President: Dr. W. E. McGarvey, 802 Jackson City Bank Bldg., Jackson.
Secretary: Dr. Don C. Lyons, 1405 Union and Peoples National Bank Bldg., Jackson.
Time: Third Thursday of alternate months.

## STATE

### Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Ralph W. Danielson, 258 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

### Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. M. H. Merriman, 115 Prospect St., Waterbury.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

### Eye, Ear, Nose and Throat Club of Georgia

President: Dr. W. L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. W. O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

### Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. R. Gillum, 221 S. Sixth St., Terre Haute.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

### Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. Royal French, 28 S. First Ave., Marshalltown.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Douglas F. Wood, 74 S. Ninth St., Minneapolis.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. L. H. Huber, Livingston.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. H. L. Harley, 101 S. Indiana Ave., Atlantic City.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. R. T. Atkins, 4 W. Fifty-Third St., New York.
Secretary: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. L. Wicks, Valley City.
Secretary-Treasurer: Dr. W. L. Diven, National Bank Bldg., Bismarck.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. M. Hendershott, 193 Eleventh St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Frank W. Dimmitt, 195 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. McWhorter, 106 E. Earle St., Anderson.
Secretary-Treasurer: Dr. G. M. Truluck, Orangeburg.
Time: April and December.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas, Texas.
Secretary: Dr. A. F. Clark, 1034 Nix Professional Bldg., San Antonio.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. D. W. Henderson, 75 S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welch, 14 Exchange Pl., Salt Lake City.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. C. P. Jones, Newport News.
Secretary-Treasurer: Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. G. A. Smith, Montgomery.
Secretary: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.

## LOCAL

### Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Charles W. Buvinger, 50 Washington St., East Orange.
Secretary: Dr. Earl LeRoy Wood, 192 Roseville Ave., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

### Academy of Medicine of Toronto, Section of Ophthalmology

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

### Atlanta Eye, Ear, Nose and Throat Society

President: Dr. L. C. Rouglin, 127 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

### Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

### Brooklyn Ophthalmological Society

President: Dr. John N. Evans, 23 Schermerhorn St., Brooklyn.
Secretary: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

### Buffalo Ophthalmologic Club

President: Dr. Joseph C. O'Gorman, 1324 Jefferson Ave., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

### Chicago Ophthalmological Society

President: Dr. Frank Brawley, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday of each month from October to May.

### Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

### Cleveland Ophthalmological Club

Chairman: Dr. Roy B. Metz, Hanna Bldg., Cleveland.
Secretary: Dr. Paul G. Moore, Medical Arts Bldg., Cleveland.

### College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. L. F. Appleman, 308 S. Sixteenth St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

### Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

### Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

### Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. Cecil C. Jones, 1121 Equitable Bldg., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

### Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

### Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. F. M. Sulzman, 1831 Fifth Ave., Troy.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

### Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

### Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

### Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. L. W. Raney, 609 Fannin St., Houston, Texas.
Secretary: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

### Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. B. J. Larkin, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

### Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

### Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 312 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

### Los Angeles County Medical Society, Section on Ophthalmology and Oto-Laryngology

President: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Mary Louise Cafe. Time: 6:30 p. m., fourth Monday of each month from October to May, inclusive.

### Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary Dr. G. Victor Simpson, Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

### Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month. Annual meeting, Jan. 10, 1933.

### Milwaukee Oto-Ophthalmic Society

President: Dr. T. F. McCormick, 324 Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

### Montreal Ophthalmological Society

President: Dr. J. P. E. Bousquet, 66 Sherbrooke St., West, Montreal, Canada.
Secretary: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

### Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

### New Orleans Ophthalmological and Otolaryngological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

### New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Algernon B. Reese, 73 E. Seventy-First St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

### Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. William P. Haney, 107 S. Seventeenth St., Omaha.
Vice President: Dr. L. G. Howard, 532 First Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: University Club, 1914 Harney St., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

### Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

### Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

### Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

### Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

### St. Louis Ophthalmic Society

President: Dr. Julius H. Gross, Carleton Bldg., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

### San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Scott C. Applewhite, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

### San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert C. Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Third Tuesday of every month except May, June, July and December.

### Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. L. Scales, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

### Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. David Hartin, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. W. W. Henderson, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

### Syracuse Eye, Ear, Nose and Throat Society

President: Dr. M. G. Brown, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

### Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy Dunlap, 108 W. Sixth St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. Sixth St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.